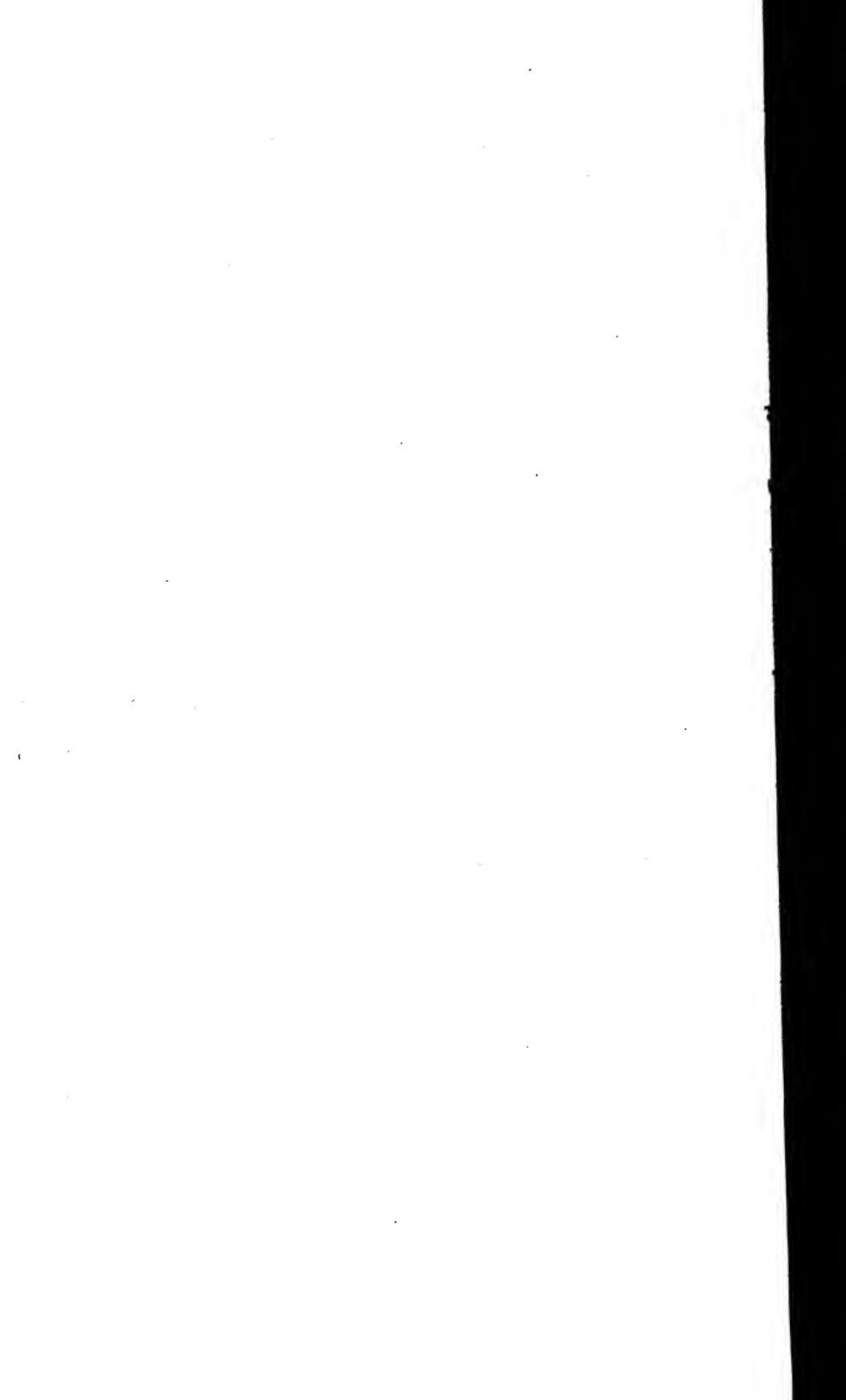
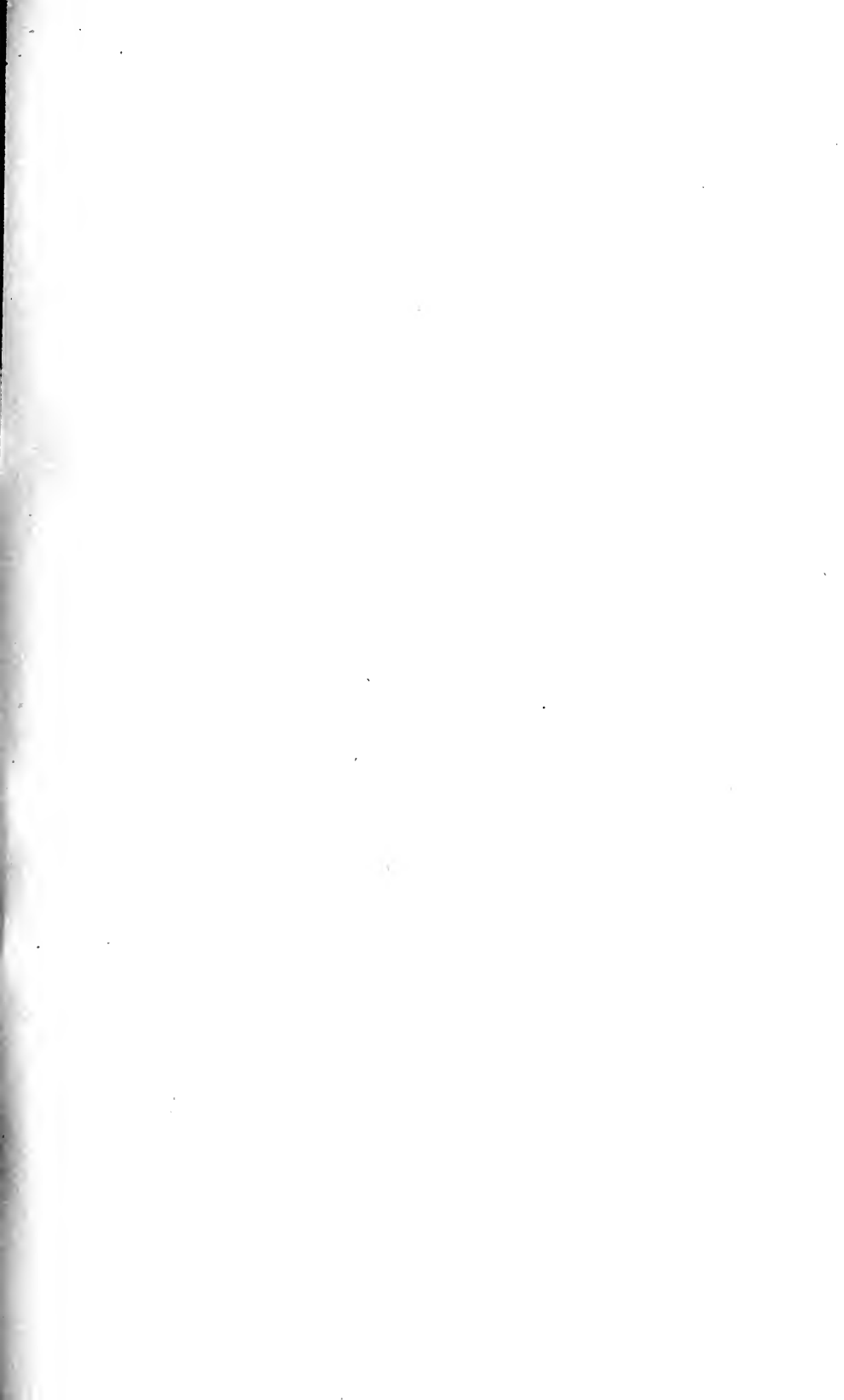


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OF

# OPHTHALMOLOGY

*FOUNDED IN 1869 BY*

DR. HERMAN KNAPP

*EDITED IN ENGLISH AND GERMAN*

BY

DR. ARNOLD KNAPP

OF NEW YORK

AND

DR. C. HESS

OF MUNICH

AND

DR. W. A. HOLDEN

OF NEW YORK

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VOLUME XLVII

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447398  
31. 5. 46

NEW YORK

G. P. PUTNAM'S SONS, 2, 4, & 6 WEST 45TH STREET

AND NEW ROCHELLE, N. Y.

LONDON: 24 BEDFORD STREET, STRAND

WIESBADEN: J. F. BERGMANN'S Verlag

PARIS: J. B. BAILLIÈRE, 19 Rue Hautefeuille

1918

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ILLUSTRATING DR. URIBE-TRONCOSO'S ARTICLE ON "OCULAR CHANGES IN ACNE  
ROSACEA. ONE CASE OF ROSACEA OCULARIS COMPLICATED  
WITH FASCICULAR KERATITIS."



FIG. 1.



FIG. 2.

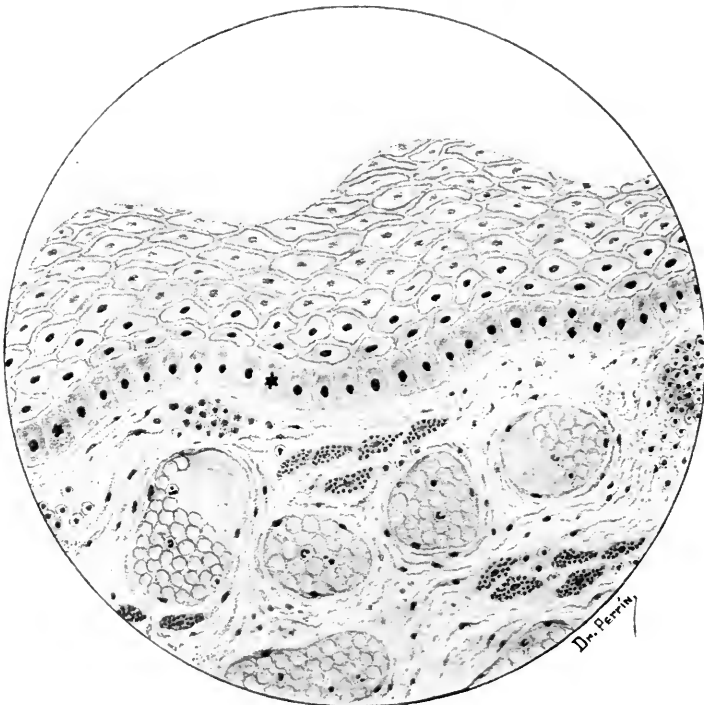


FIG. 3.

Dr. Parrin

## ARCHIVES OF OPHTHALMOLOGY.

OCULAR CHANGES IN ACNE ROSACEA. ONE CASE  
OF ROSACEA OCULARIS COMPLICATED  
WITH FASCICULAR KERATITIS.

BY DR. M. URIBE-TRONCOSO.

ASSISTANT PROFESSOR OF OPHTHALMOLOGY, NEW YORK POST-GRADUATE  
MEDICAL SCHOOL AND HOSPITAL, NEW YORK.*(With three illustrations on Text-Plate I.)*

THE ocular localizations of acne rosacea have been studied with great interest in late years. Although its existence was pointed out by Arlt in 1864 and afterwards described by Fuchs and Vossius, the characteristic signs of the disease were only definitely stated by Capauner (1), Kuntz (2), Wich-erckiewicz (3), Schirmer (4), Blancke (5), Sydney Stephenson (6), and later by Erdmann (7), Holloway (8), Caralt (9), and others (10).

Erdmann proposed the names of Rosacea Keratitis and Rosacea Conjunctivitis, which have been adopted, and accordingly this disease is so described in modern text-books and monographs, but these names in our opinion are limited and must be substituted by the more comprehensive term of Rosacea Ocularis.

Although numerous cases of this disease have been published in recent years, the clinical aspects of the varied manifestations of rosacea of the eye have not yet been described, nor its etiology and pathogenesis entirely proved. All authors have mentioned the great resemblance of the ocular rosacea to

phlyctenular keratitis, but until recently no reference of its association with fascicular keratitis has been recorded. For this reason, as I was fortunate enough to observe a case of this nature, I think it worth while to describe and discuss it thoroughly in order to ascertain the character of the disease.

The history of the case is as follows:

A. B., 44 years old, was first seen by me on September 9, 1914. He is a strong and vigorous farmer, who began to notice some reddening of the right eye about a year previously, produced according to his own statement by the introduction of a foreign body on the conjunctiva.

He gave a history of gonorrhœa and chancroid at the age of twenty-one and chronic nasal catarrh. No evidences of syphilis were found. He was a heavy drinker of wines and liquors until lately, when he stopped alcohol.

Since youth he had on both cheeks red plaques of rosacea with which he was very pleased because these gave him an appearance of health, but one year ago the red areas became irritated and an attack of acne occurred extending gradually afterwards to the forehead.

Some weeks after the beginning of the acne the right eye was involved in the inflammation, which, according to the patient's statement, was confined to the inner portion of the conjunctiva. In May, 1914, after prolonged exposure to the night air the skin of the face became more inflamed and the injection of the conjunctiva extended to the cornea where a white spot appeared, accompanied by photophobia and lacrymation. In July of the same year another white spot appeared in the outer part of the cornea, with increased irritation and lacrymation. As his condition became worse he was compelled to come to the city for consultation.

His condition when I saw him was as follows: On both cheeks there existed extensive and highly congested areas of acne rosacea, covered with a great number of small pustules of acne and suppurative folliculitis, much more confluent near the lower eyelid. The naso-malar furrows were smeared with the greasy secretion characteristic of seborrhea.

On the right eye the skin and margins of the lids were normal, but a marked blepharospasm was present and a great quantity of tear secretion flushed out when the lids were forcibly separated. The conjunctiva was strongly injected. Upon the inner portion of the cornea could be observed, advancing almost to its middle third, one stripe of dense gray-white infiltration ending in a broader white



crescent (Fig. 1). On the surface of the stripe ran many blood-vessels coming from the conjunctiva and terminating at the base of the white crescent. All around the fascicle and in the internal half of the limbus there existed a faint dotted zone of infiltration, 2mm broad. Under the fascicle there was a small white circular subepithelial spot, about the size of a pin head. The pericorneal injection was very marked on the inner aspect of the limbus and advanced upon the faint rim on the cornea. The episcleral tissue at the internal canthus was also very red and inflamed.

In the outer and lower quadrant of the cornea another band of infiltration existed, which extended less than the internal and ended also in a larger gray-white crescent. The injection was much less at the external portion of the limbus and there was no trace of infiltration rim.

The left eye was normal.

Photophobia and blepharospasm subsided rapidly under the use of holocaine. Antiseptic wash, atropine, and hot compresses were instituted with good results. For the treatment of the skin condition the patient was referred to the dermatologist, who prescribed the proper internal and external medicaments.

With this treatment the outbreak of acne and the congestion of the skin subsided. At the same time the condition of the eye improved rapidly. The conjunctival injection subsided and the zone of faint infiltration around the fascicle and in the limbus became less noticeable.

On September 15th no photophobia or lacrymation was apparent. The internal fascicle became less vascular, did not progress further, and was more white and dense. The faint rim of infiltration and the white spot below have disappeared. The external fascicle remained in the same condition.

On September 20th, coincident with a new congestive attack of acne pustulosa in the skin, the eye became greatly irritated. Photophobia and lacrymation returned and the external fascicle progressed toward the center of the cornea by the enlargement of the crescent, which assumed a triangular form and stained slightly with fluorescein (Fig. 2). The vascular pencil is lying in a depressed, transparent furrow, which mirrors with the reflected images.

On September 22d there appeared upon the scleral conjunctiva at the external angle, an inflammatory nodule yellowish-white in color and of the size of a millet grain, sharply outlined and elevated upon the surrounding conjunctiva. Numerous blood-vessels converged to it from all directions. Two days afterwards the congestion and irritation of the skin diminished and the condition of the

eye improved. Cicatrization continued toward the internal fascicle, whose vessels diminished in number. The external fascicle on the contrary progressed steadily and its apex became larger. Powdered iodoform and a protective bandage were prescribed. On September 29th the outbreak of acne on the face terminated. The congestion of the plaques of rosacea subsides and desquamation begins. On the eye the bulbar conjunctiva recovered its normal color at the internal angle. Outside the episcleral nodule increased until it became the size of a wheat grain and its local vascularization became more marked. This nodule is firmly adherent to the sclerotic and the conjunctiva does not slide upon it. The internal fascicle is now entirely cicatrized; the blood-vessels become absorbed almost completely, leaving a white opaque band. The external fascicle appears to be stationary and the apex is less infiltrated.

Patient does not return for consultation until the 28th of October, having been compelled to go to his farm on important business for some days. During his absence, it appears, he continued treatment instituted for the eye, but neglected the skin condition, and a new outbreak of congestive acne came back. The eye is almost quiet; the scleral nodule is in the same condition. The vascular fascicle outside has not progressed, its triangular apex has disappeared, and the vessels run to its end.

In order to study the histological characters of the scleral nodule I performed the excision of a small piece under cocaine anæsthesia. The treatment of the skin condition was resumed and eight days afterwards the acne disappeared and the plaques of rosacea were much discolored. In the eye the episcleral nodule became small and finally disappeared.

At this stage cicatrization was rapidly progressing at the external fascicle and became completed in eight days, leaving a characteristic white leucoma, which almost reaches the pupillary margin although it is smaller than the internal stripe.

The patient is discharged with special caution not to abandon the treatment instituted for the skin condition. The histological examination of the excised nodule was made by Dr. Tomás G. Perrin, who gave the following account:

"The specimen sent for examination consisted of epithelium and dermis of the bulbar conjunctiva and part of the loose connective tissue of the episclera (Fig. 3).

"*Epithelium*. Presents the stratified pavement structure characteristic of the regions far away from the fornix. This stratification is notably increased by an hyperplastic

process, which has retained the normal orientation of the cells and the subepithelial basal membrane. Neoplastic lesions are not observed. In none of the numerous sections studied were there found any traces of acinous glandular formations (Krause's or Wolfring-Ciaccio's) nor tubular (or Henle's) nor utricular (or Manz's).

"*Dermis.* Rudimentary papillæ are visible on the sections. Disseminated on the conjunctival stroma there are numerous foci of infiltration, formed by lymphoid cells and lines of plasma cells. There are numerous blood-vessels, some of them newly formed with embryonic walls, which present congestive lesions and slight dilatations, which cannot be considered as a true telangiectatic condition. There is a considerable increase in fibroblasts with hypergenesis of connective tissue fibers, principally localized around the vessels."

Describing the symptomatology of the ocular rosacea all authors have pointed out the striking resemblance between rosacea keratitis and phlyctenular kerato-conjunctivitis. Although in the case I have just described it was not possible to observe the beginning of the keratitic inflammation, it is almost certain that it began with efflorescences or infiltrations at the limbus which became ulcerated and afterwards acquired a serpiginous character, with tendency to invade the center of the cornea. The patient himself clearly stated that "a white spot" was at first formed on the eye and then progressed toward the pupil. I was able to observe the extension upon the cornea of the external stripe, the brush of vessels trailing after the ulcer, and then the characteristic leucomas, which leave no doubt of the nature of the keratitis, a true fascicular keratitis complicating an acne rosacea of the skin.

According to Seo and Yamaguchi the vessels advance below Bowman's membrane, which is destroyed behind them, and sometimes they invade also the most superficial layers of the corneal stroma. As the vascular brush advances it is placed in a groove and the epithelium regenerating above it is easy to detect a semi-transparent furrow which mirrors on the reflected light and ends in a gray-white, triangular or crescent-shaped zone of infiltration whose border is slightly stained by fluoresceine.

The internal infiltration existed probably about five months

before I saw the case. It became ulcerated again on the second attack, when he came to my office.

It is important to state that in this patient, contrary to the opinions of several authors, the ocular changes were intimately associated in their progress with the skin condition and that the improvement of the latter brought always a relief of the irritation and congestion of the eye, although, of course, the advance of the fasciculus was not checked.

Keratitis fascicularis has never been mentioned as a complication of rosacea ocularis. Its presence in this patient confirms the close relationship between phlyctenular keratitis and rosacea keratitis, because *the fasciculus has never been found associated with any other condition than phlyctenular keratitis.*

The ocular manifestations of acne rosacea, according to several authors, are as follows: In the *cornea* the lesions consist of gray-white infiltrations limited or diffuse, subepithelial, either similar to those of phlyctenular keratitis or much larger and deep, parenchymatous (Holloway, Capauner), surrounded by faint punctate zones of infiltration, which may progress by successive outbreaks, recurring readily and sometimes being covered with abundant blood-vessels (although less marked than in pannus phlyctenularis). Upon these infiltrations small superficial ulcers develop, which may sometimes be infected or acquire a serpiginous character. Erdmann has described a case of *ulcus rodens* (Mooren's). Blancke observed an ulcer which became wider and deep and was accompanied by hypopyon.

Corneal ulcers leave after healing flat or more commonly excavated scars which easily become reinfected during new outbreaks of ocular inflammation, coincident with inflammatory attacks on the skin. Sometimes the entire cornea may be opacified (Capauner).

The *iris* may become involved in some cases: hyperæmia only or true iritis (Caralt, Blancke).

In the *sclera* there is a formation of nodules or papules of variable size, which generally undergo absorption, and disappear without any sequelæ.

Rosacea keratitis differs clinically from keratitis phlyctenularis by the presence of the faint dotted infiltration area around

the corneal lesions, by the extension of the changes, its abundant vascularization, its preference for the inferior quadrant of the cornea, the complication with iritis, and the excavated form of the scars which result from corneal ulcers. But these clinical distinctions should not be sufficient to make the differential diagnosis, were it not for the existence of the skin changes, the rosacea and acne, and if the age of the patient did not exclude a true phlyctenular keratitis.

Let us consider what we know of the etiology of this last disease and ascertain if something similar is responsible for the appearance of ocular rosacea. We do not know definitely what is the cause which produces phlyctenular kerato-conjunctivitis. Is it an ectogenous or an endogenous infection? Is it a toxæmia or a toxi-infection? Bacteriological researches have proved that the efflorescence itself is sterile when recent. Axenfeld (11) long ago opposed the views of Bach, who considered staphylococcus as the cause of the infection, and asserts that this microbe being a normal host of the skin, only secondarily infects the conjunctiva.

More promising were the researches on the endotoxic origin of the phlyctenular keratitis. Many authors: Derby, D'Ayrens, Igersheimer, Weekers, Stephenson, Rosenhauch, and others have obtained with the general tuberculin test from 50% to 90% of positive reactions. Diagnostic injections of tuberculin sometimes produce an eruption of phlyctenes in the eye (Schutz and Videky, Feer, Stock, etc.). The old ophthalmoreaction proved itself positive not only in the form of conjunctivitis, but also as conjunctival phlyctenes and even as a keratitis.

Experimental researches made by Rosenhauch (12) have proved that in rabbits infected with tuberculosis, conjunctival phlyctenes can be produced by instilling in the conjunctival sac pure cultures of staphylococcus aureus, the conjunctiva being previously irritated; but he failed in obtaining them in normal rabbits by the irritation of the conjunctiva and the use of staphylococcus cultures. Weekers (13) was able to produce also phlyctenules on the conjunctiva of tuberculous rabbits by the instillation of tuberculin into the eye, and asserts that the external action of the staphylococcus is not necessary when the tuberculous toxæmia exists.

Lately Rubert (14), repeating the experiments of Rosenhauch and Weekers, has confirmed the conclusions of the latter in regard to the positive effect of the instillation of tuberculin, but adds that phlyctenules can also be produced by the action of the toxins of staphylococcus aureus on the eye, it being always necessary in this latter case that a previous general tubercular infection exists.

These experiments seem to confirm the idea of the scrofular or rather tubercular origin of the eye changes in phlyctenular kerato-conjunctivitis, and in fact in a great number of children that react to tuberculin, clinical evidences of tuberculosis can be found. But there are undoubtedly many other cases in which the patient's general health seems to be normal and no traces of scrofulosis can be found. In these cases, however, general autointoxication by faulty diet and intestinal disorders or some local infection as nasal catarrh or adenoids are at fault.

There are other children in whom without clinical evidences of tuberculosis the ocular efflorescences are accompanied by eczema of the face and the other signs of Czerny's "exudative diathesis." According to our present knowledge therefore two factors seem to be necessary for the production of eye phlyctenes: 1st. A general toxæmia by the tuberculous bacilli or some autointoxication specially of intestinal origin. 2d. An irritative local agent which may be the staphylococcus or the tubercle bacillus or perhaps the streptococcus in some cases, as suggested by Saboureaud.

The same factors account for the development of phlyctenules in the case of rosacea ocularis? We must certainly discard tuberculous toxæmia as the cause of rosacea in the eye. There is no relation between them. But we probably have at the bottom of the ocular rosacea a toxæmia either of intestinal or, as certain authors suggest, of genital origin.

Let us consider in what way the two characteristic elements of acne rosacea work upon the membranes of the eye.

There is not a single case recorded of *simple rosacea*, that is of congestive or telangiectasic patches on the face without acne, which had been accompanied with ocular manifestations. In all recorded cases there was always infection of the sebace-

ous glands, *i. e.* inflammatory acne, which had certainly been preceded by cutaneous seborrhea.

On the other hand acne polymorphus alone, without rosacea, when it settles in the skin of the face never gives rise to conjunctival or corneal lesions. This is easily explained, as the acne being due to a special infection of the sebaceous glands of the skin, which begins with seborrhea and sometimes is attended with secondary staphylococcus infection of the gland and follicle, cannot be started in the conjunctiva in which there are no sebaceous glands. Therefore if the ocular manifestations do not exist when the two elements of acne rosacea are separated and only come up when these elements are combined, its appearance must be due to the fusion of their respective etiological factors.

The rosacea plaque itself has been attributed to several factors. Some authors claim it is purely mechanical, and produced by the compression of the veins in the cranial foramina. Others ascribe it to a parietic condition of the vascular walls, very closely allied to the complex factors which produce varicosities of the lower extremities. The Vienna school supports the theory that it is *angio-neurotic* in origin, and its cause must be looked for in all processes which, directly or by the intervening action of the centers and vasomotor nerves which govern the circulation of the blood in the face, give rise to the dilatation of the capillaries and small veins in the skin. Among these processes are to be mentioned the repeated congestion of the face which is produced after copious meals in persons whose digestion is imperfect and who generally have cold extremities and hot face. In its ultimate development gastro-intestinal and hepatic troubles are much to be blamed, and many assert also that menstrual disturbances and exaggerated continence play some rôle. But all these factors are impotent to produce true acne rosacea if the skin is intact and seborrhea does not exist. On the other hand when the sebaceous glands become infected with the microbacillus seborrheicus, if the patient suffers from gastro-intestinal troubles and autointoxication occurs, it is almost certain that paroxysmal outbreaks of discrete acne pustules, first of small size and then larger, will come on the plaques of rosacea. These pustules are produced by the late infection

of the seborrheic glands and orifices of the hair follicles with the staphylococcus. Therefore, it is probable that the production of ocular manifestations in acne rosacea may be due to the existence of a general toxæmia and to the action of some external irritant as the toxins of the staphylococcus and perhaps of other microorganisms existing in the skin, which are conveyed to the eye by soiled hands, dirty handkerchiefs, etc.

Ocular rosacea is frequently accompanied by nasal troubles. Stellwagon has called attention to its coincidence with certain diseases which give rise to obstruction of the nasal fossæ such as polypi, hypertrophy of the head of the median turbinate, or hypertrophic rhinitis. The infection of the nasal mucous membrane by the streptococcus is frequent in childhood and in adults an infection by the staphylococcus exist sometimes.

Many authors consider the acne rosacea ocularis to be due to nervous or trophic changes. Holloway ascribed the disease to a nervous lesion localized in the territory of distribution of the fifth nerve, specially in the maxillary branch. Burton Chance (15) suggests that there are trophic changes in the terminal nerves of the cornea. Verhoeff believes that the corneal lesions are of neuropathic origin and due to impulses of the Gasserian ganglion passing along the conjunctival nerves, these lesions being of the same origin as certain small peripheral corneal infiltrations associated with herpes facialis. However he has never been able to demonstrate any reduction in the sensibility of the cornea in the periphery. Caralt (16), who has made a detailed study of ten cases of rosacea ocularis (in the other four cases he mentions there were no ocular lesions), believes that the ocular changes depend on an angio-neurotic process, reflex in origin, similar to that of the skin and produced by autointoxications which are due either to chronic diseases of the digestive apparatus or to functional disturbances of the glands of internal secretion of the genital apparatus. For him ocular rosacea is not of bacterial origin but simply an endotoxic condition, which must be separated from the phlyctenular keratitis. However he never found—except in one of his cases—anæsthesia of the conjunctiva or cornea nor has he ever observed eruption of vesicles along the branches of the trigeminus.



We must remember on this account that latest researches have found the Gasserian ganglion infected by some blood-carried bacteria in cases of neuritis.

In regard to the pathogenesis of fascicular keratitis we can say nothing in concrete. Addario (17), who has examined bacteriologically twelve patients affected with this complication, has found in all, except one, the staphylococcus, but this bacterium is by no means specific; it is found also, as we have said, in the efflorescence which never becomes serpiginous.

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## THE PRODUCTION OF CATARACT.

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(From experiments carried out at Nela Research Laboratory.)

*(With seven figures on Text-Plates II-III.)*

IT is recognized that cataract is of more frequent occurrence among people living in the tropics, glass-blowers, diabetics, and elderly persons than among people generally. Some attribute the prevalence of cataract in the tropics to the excess of ultra-violet radiation in tropical light, while others attribute it to the heat. That ultra-violet plays a part in the production of cataract in the tropics is rendered very probable by the fact that cataract in the tropics as a rule begins in the lower quadrant of the lens which is exposed to the light coming directly from the sky, and hence richer in ultra-violet radiation than the light reflected from the ground, to which the upper quadrant of the lens is exposed. The prevalence of cataract among glass-blowers is usually considered to be due to the extreme heat to which the eyes of the glass-blowers are exposed. The prevalence among diabetics is explained on the assumption that the abnormal amount of sugar in the liquids of the body extracts water from the lens, thus rendering it opaque or cataractous. The prevalence among elderly people is attributed to the fact that the lens continues to increase in size throughout life, hence, with advance in years the nucleus of the lens is further and further removed from the source of nutrition, and this condition, it is claimed, predisposes to nuclear opacity. If these generally accepted causes for cataract are examined critically, certain questions arise as, for example, how is it possible for the temperature of the eyes of people living in the tropics or of glass-blowers to

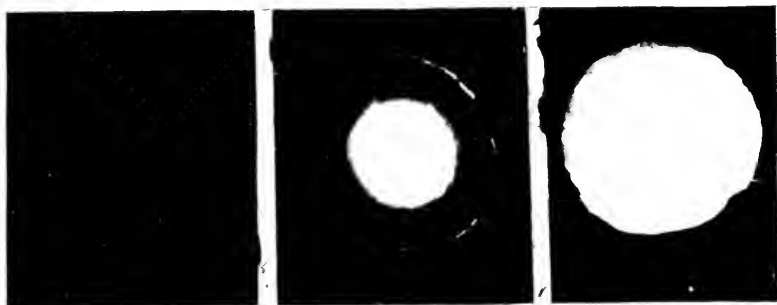


FIG. I.—Photograph of crystalline lenses. A, normal transparent lens; B and C, opaque lenses. B, nuclear opacity produced by immersion in 15 per cent. potassium chloride; C, cortical opacity produced by immersion in 5 per cent. calcium chloride.

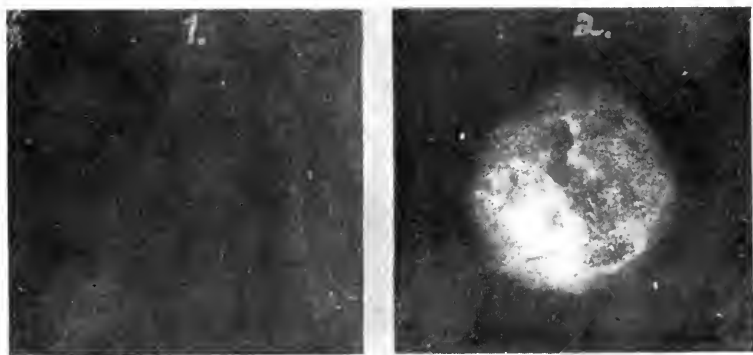


FIG. II.—(1) is a photograph of the square of glass covered with egg white, the central area of which had been exposed to ultra-violet radiation. (2) is a photograph of the same square of glass after it had been immersed in a 0.1 per cent. calcium-chloride solution.

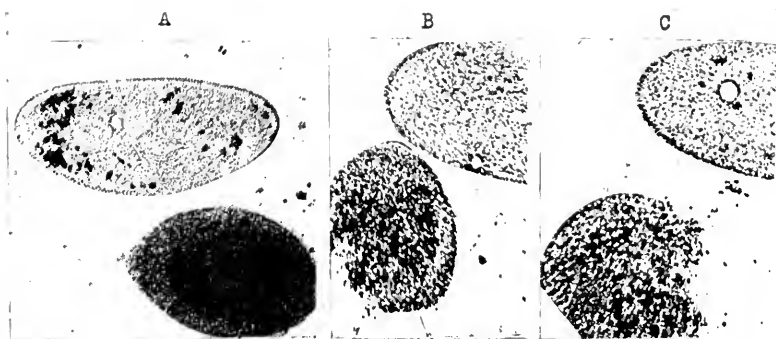


FIG. III.—Microphotographs of paramecia. The upper ones under A, B, and C are the normal transparent living animals: the lower one under A was killed by heating to 90° C.; the lower one under B, by heating to 45° C.; the lower one under C, by exposure to ultra-violet radiation.



rise sufficiently high to produce an opacity of the lens without permanent injury to other parts of the eye, or how does the deprivation of nutrition render a transparent body, such as the lens, opaque?

The most apparent change in the lens in cataract is its loss of transparency, hence, in seeking for the cause of the disease the thing to be determined is the cause of the opacity of the lens. Chemically the most conspicuous difference between cataractous and normal lenses is to be found in the inorganic constituents. In the table below are given the results of analyses (1)

TABLE I.

AVERAGE PERCENTAGE COMPOSITION OBTAINED BY ANALYZING THE COMBINED ASH OF A LARGE NUMBER OF LENSES.

	<i>Percentage Ca in Ash</i>	<i>Percentage Na in Ash</i>	<i>Percentage Si in Ash</i>
Normal adult human lens	?	?	0
Normal adult pig's lens	0.08	6.67	0
Embryo human lens	?	?	0
Cataract human lens (U. S. A.)	12.50	23.82	0
Cataract human lens (India)	6.00	25.06	3.63

of normal, as well as cataractous lenses from the United States and from the tropics (India). It may be seen that the amount of calcium and sodium salts in the cataractous lenses was increased over the normal and that there was a very appreciable amount of silicon in the cataractous lenses from India, whereas there was none in those from the United States. Silicates may possibly be accounted for in the cataractous lenses from India by the fact that silicious earths form a part of the diet of certain classes of the natives. The increase in inorganic salts in cataractous lenses suggested that the salts might be a factor in the production of cataract, hence experiments were carried out to determine if an opacity of isolated lenses could be produced by immersing them in solutions of these salts.

*Effect of Salt Solutions on Isolated Lenses.* One set of lenses was immersed in 15% sodium chloride and another set in 5% calcium chloride for fifteen hours. At the end of this time

the lenses were removed from the solutions and photographed. The photograph is shown in Figure I. A is the normal transparent lens, B the one immersed in 15% sodium chloride, C the one immersed in 5% calcium chloride. It may be seen that sodium chloride produced nuclear opacity without affecting the transparency of the cortex, and it was found upon cutting the lens in two that calcium chloride produced cortical opacity without affecting the transparency of the nucleus. It was also found that potassium chloride produced nuclear opacity without affecting the transparency of the cortex of the lens.

If the inorganic salts are involved in the production of cataract, nuclear and cortical, the sodium salts function in the production of nuclear and the calcium salts in the production of cortical cataract. The objection to the assumption, however, that either of these salts can of themselves produce cataract in a living animal is the fact that the strength of the solutions necessary to produce an opacity in the isolated lens is far stronger than could ever occur in a living animal, hence if the inorganic salts are involved at all in the production of cataract it must be conjointly with some other factor. Since the chief function of the crystalline lens is the transmission of radiant energy, the following experiments were carried out to determine if radiant energy acting alone or conjointly with these salts could produce an opacity of the lens or cataract.

*Effect of Infra-Red.* Sir William Crookes (2) concludes that glass-blower's cataract is due to the long waves of the spectrum. This conclusion was based on the fact that he found the radiation from molten glass in the glass-blower's furnace to be very rich in red and infra-red. In the present instance experiments were carried out to determine if it were possible to produce an opacity of the lens, or cataract, by the direct exposure of excised pig and ox lenses to the radiation from an electric furnace, which is particularly rich in infra-red. Lenses fitted into floats made of thin rings of cork were almost submerged in open-mouthed glass vessels containing egg-white, blood serum, aqueous and vitreous humors, respectively. Each glass vessel was placed in a tank of running water with its mouth slightly above the surface of the water. An electric furnace operating at 1000° C. was inverted and

## ILLUSTRATING DR. BURGE'S ARTICLE ON "THE PRODUCTION OF CATARACT."

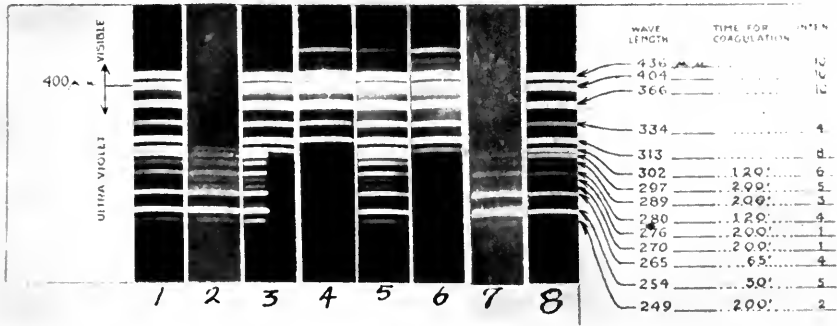


FIG. IV.—Photograph of spectra of small quartz mercury-vapor burner. (1) made on photographic plate; (2) made on lens protein; (3) made on photographic plate one-half of the slit of the spectrograph being covered with the cornea of a rabbit; (4) through layer of lens protein 1mm thick; (5) through aqueous humor 1mm thick; (6) photograph of spectrum made on photographic plate; (7) made on egg white; (8) made on photographic plate through egg white 1mm thick.

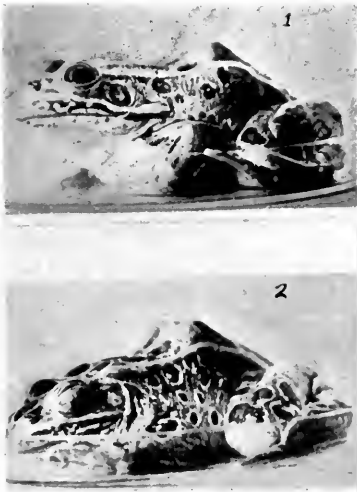


FIG. V.—Frog (1) living in tap water and exposed to ultra-violet radiation for 5 hours. Frog (2) living in 0.2 per cent. sodium-silicate and exposed to ultra-violet radiation for 5 hours.

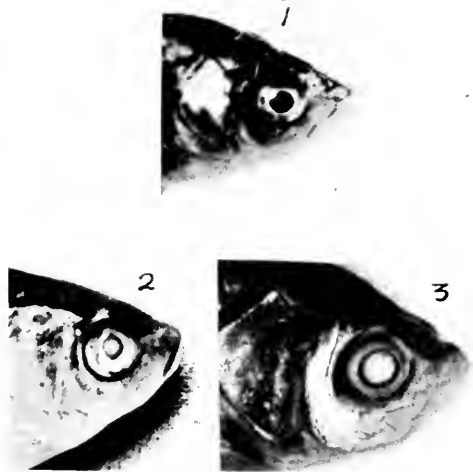


FIG. VI.—Fish (1) living in tap water and exposed to ultra-violet radiation for 12 hours. Fish (2) living in 0.1 per cent. sodium silicate and exposed to ultra-violet radiation for 12 hours. Fish (3) living in 0.1 sodium silicate and exposed to ultra-violet radiation for 24 hours.

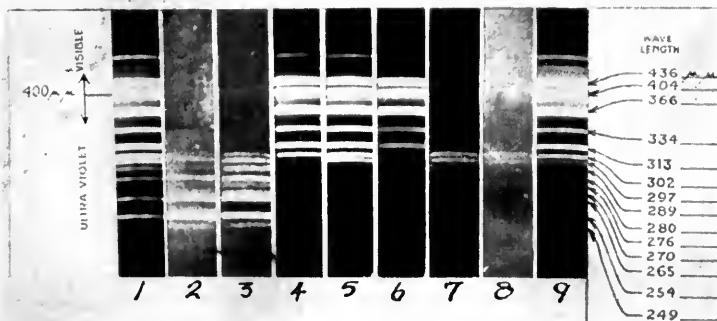


FIG. VII.—Photograph of spectra of the small quartz mercury-vapor burner and of the large quartz mercury-vapor burner. (1) that of the small burner; (9) that of the large burner, made on a photographic plate. (2) that of the

small burner; (8) that of the large burner, made on egg white. (3) that of the small burner; (7) that of the large burner, made on lens protein extracted with 0.1 per cent. calcium-chloride solution. (4) that of the large burner made through cornea of the rabbit; (5) made through glass 1mm thick; (6) through glass 5mm thick.





placed directly over the lenses at a distance of 15 $cm$ . The materials were thus exposed for many hours without the production of an opacity either in the lenses or in the media. Other experiments were carried out similar to those just described, except that the furnace was placed 5 $cm$  from the surface of the lenses. Under these conditions the lenses could be rendered opaque in about ten minutes. However, on placing the bulb of a thermometer on a lens thus rendered opaque it was found that the temperature in the region of the lens was about 80° C. Hence the opacity thus produced was evidently due to the heat effect and not to the red and infra-red radiation.

*Effect of the Visible Spectrum.* Lenses were arranged on floats similar to those described above and almost completely immersed in similar media, viz., egg-white, blood serum, aqueous and vitreous humors. By means of a bi-convex glass lens 10 $cm$  in diameter and with a principal focal distance of approximately 30 $cm$  the filament of a nitrogen-filled tungsten lamp operating at 2000 cp. was focused on the lenses. Exposures for many hours were made. In no case was an opacity of the lenses produced.

Similar experiments were made by focusing the image of the sun on the lenses. Opacity in this manner could be produced in a very few minutes, but it was found in every case that the temperature of the crystalline lens was raised sufficiently high to coagulate its protein.

The conclusion is therefore drawn that an opacity of the lens, or cataract, cannot be produced by radiation from the region of the visible spectrum or of the infra-red provided the temperature effect be excluded.

*Effect of Ultra-Violet Radiation.* Schanz and Stockhausen (3) studied the radiation from the glass-blower's furnace and found, contrary to the findings of Crookes, that it was rich in ultra-violet radiation, hence they concluded that glass-blower's cataract is due to the short waves.

Experiments were devised by the writer to determine if it were possible to produce an opacity in the crystalline lens by direct exposure to the radiation from the quartz mercury vapor arc. Lenses were introduced into quartz tubes previously filled with egg-white, blood serum, aqueous and

vitreous humors, respectively. The diameter of these quartz tubes was slightly less than the diameter of the lenses, so that each tube was filled in an horizontal direction by means of the lens. The tubes were closed with rubber stoppers and placed horizontally in a tank of running water at a depth of 1cm beneath the surface of the water and 5cm below a quartz mercury-vapor burner operating at 140 volts, 3.3 amperes and about 2500 cp. The egg-white and the blood serum were firmly coagulated after twenty hours' exposure, while the aqueous and vitreous humors had become turbid. The immersed lenses in all the tubes, however, were as transparent after such an exposure as they were at the beginning of the experiment. At the end of one hundred hours' exposure there was practically no change in the transparency of the lenses. Other experiments were carried out exposing egg-white, blood serum, aqueous and vitreous humors in the quartz tubes without the immersed lenses. At the end of twenty hours' exposure the egg-white and the blood serum were coagulated, but the transparency of the aqueous and the vitreous humors was affected very little, if any, by such an exposure. These exposures were continued for one hundred hours, and at the end of this period the aqueous humor was as clear as it was at the beginning of the experiment, while the vitreous humor had been rendered very slightly cloudy.

The conclusion is therefore drawn that ultra-violet radiation which is of sufficient intensity to render egg-white and blood serum firm coagula in a few hours will not affect the transparency of the crystalline lens or of the aqueous and vitreous humors in one hundred hours' exposure. Dreyer and Hanssen (4) found that ultra-violet radiation will precipitate not only egg-white and blood serum, but practically every other protein and related substance. Thus it would appear that the lens protein and the proteins of the aqueous and vitreous humors form a conspicuous exception in that it is practically impossible to coagulate these by means of ultra-violet radiation, under similar conditions.

*Effect of Infra-Red and Visible Spectrum on Lenses in Salt Solutions.* Lenses were immersed for two hours in 0.1% calcium chloride, 0.1% magnesium chloride, 0.1% sodium silicate, and 1.0% dextrose, respectively. They were then

exposed to the radiation from the electric furnace, the tungsten nitrogen-filled lamp, and the sun. Exposures were made as in the preceding experiments without affecting the transparency of the lenses or of the media, provided the heat effect was excluded.

*Effect of Ultra-Violet Radiation on Lenses in Salt Solutions.*

In the same manner, quartz tubes containing similar media and immersed lenses were prepared and exposed to the radiation from the quartz mercury-vapor burner. It was found that the solution in the quartz tube containing a lens immersed in the 0.1% calcium chloride became turbid after twenty-five minutes' exposure. The portion of the lens on the side next to the burner had become an opaque mass after seventy-two hours' exposure, whereas the transparency of the half of the lens away from the burner was only slightly affected. The same thing happened on the exposure of the lenses immersed in 0.1% magnesium chloride, in 0.1% sodium silicate, or 1.0% dextrose, except that the effect was not so marked and was not produced so quickly as when calcium chloride was used.

The conclusion is therefore drawn that ultra-violet radiation cannot precipitate lens protein in its native state but that it can precipitate it and hence produce an opacity or cataract when the lenses are immersed in solutions of those salts found to be greatly increased in cataractous lenses. The following experiments were carried out to show that the action of ultra-violet radiation, in precipitating lens protein consists in modifying it so that the salts can combine with the protein to form a coagulum.

*Effect of Ultra-Violet Radiation on Proteins.* Egg-white was poured on a glass plate 6cm square and permitted to dry. A piece of cardboard the size of the plate with a circle cut from its center approximately 2cm in diameter was fitted over the plate. The preparation was exposed for thirty hours to the radiation from the quartz mercury-vapor burner at a distance of 10cm. It will be noted that only the central circular area of the egg-white was exposed to the radiation since the peripheral portion was covered by the cardboard. At the end of the thirty hours' exposure the cardboard was removed and the plate was photographed. Figure II. (1) is the photograph. It may be seen that at this time there was no apparent differ-

ence between the exposed central area and the unexposed peripheral area of the layer of egg-white. The plate was then immersed in a 0.1% calcium chloride solution. (2) is a photograph of the plate after it had been immersed in the solution for one hour. It may be seen that the calcium chloride precipitated the proteins of the egg-white in the circular area where it had been exposed to the radiation, while it did not affect the egg-white where it had not been exposed. Calcium nitrate was tried in the same manner and found to have a similar effect in precipitating the protein of the egg-white previously exposed to the radiation.

A thin layer of lens material was made by pressing three fresh pig lenses between two quartz plates. The cardboard with a circular area cut from its center was placed over the preparation and this was exposed to the radiation from the quartz mercury-vapor burner for thirty hours as the egg-white had been. At the end of this time there was no apparent difference between the exposed circular area and the unexposed peripheral area of the lens material, both being transparent. When, however, the preparation was immersed for about an hour in 0.1% calcium chloride solution the exposed circular area became an opaque mass, while the unexposed peripheral area remained transparent. The conclusion may be drawn that ultra-violet radiation coagulates protein by changing it in such a way that salts such as those of calcium can combine with it to form a coagulum. In this respect it would seem that ultra-violet radiation acts on protein in very much the same way that certain enzymes act on it. It is known, for example, that rennin changes the protein in milk, caseinogen, in such a way that calcium salts can combine with it to form an insoluble curd, casein.

*Further Analysis of the Action of Ultra-Violet Radiation.* The following experiments were carried out to show that ultra-violet radiation not only changes proteins so that certain salts can combine with them to form a precipitate, but that it also changes the staining property of the protein and that the change produced in the protein is molecular in character and not atomic.

A thin film of egg-white was poured on a glass plate and permitted to dry. A piece of cardboard the size of the plate

with a circle cut from its center approximately 2cm in diameter was fitted over the plate. The preparation was exposed for twenty hours to the radiation from the quartz mercury-vapor burner at a distance of 10cm. It will be noted that only the central circular area of the egg-white was exposed to the radiation, since the peripheral portion was covered by the cardboard. At the end of the twenty hours' exposure the cardboard was removed and the plate was immersed in a weak solution of an aniline dye, rhodamin. The central circular area that had been exposed to the radiation was intensely dyed, whereas the unexposed peripheral area did not take the dye. A thin layer of the crystalline lens was tried in a similar manner with the same result that was obtained in using egg-white.

The conductivity of a thin layer of egg-white as well as of lens material was determined before being exposed to ultra-violet radiation, during the exposure and after it. Precautions were taken to avoid evaporation and to maintain a constant temperature during the exposure. It was found that the exposure did not alter the conductivity, hence it is evident that the change produced in protein by the short wave-lengths of the spectrum so that certain salts can combine with it to form a precipitate is molecular in character and not atomic.

*The Action of Ultra-Violet Radiation in Killing Living Cells.* The following experiments present evidence that ultra-violet radiation acts on living cells in the same manner that it acts on fresh dead cells, and that the destructive effect of ultra-violet radiation is due to the coagulation of the protoplasm of the living cells. Several drops of water containing great numbers of unicellular organisms, paramecia, were introduced into a shallow glass vessel and covered with a quartz plate. The glass vessel was then placed on a block of ice under a quartz mercury-vapor burner operating at 140 volts and 3.3 amperes, at a distance of 5cm, and in this position the organisms were exposed for twenty minutes. A drop of the liquid containing the dead paramecia was mixed with a drop containing living ones on a glass plate and covered with a cover glass. Having located under a microscope a dead organism and a living one lying close together a micro-photograph was made of them. Similarly, micro-photographs were made of living

paramecia and of paramecia killed by heating to 45° C. and 90° C. respectively. These photographs are shown in Figure III. The upper organisms under A, B, and C are living transparent animals; the lower one under A was killed by heating to 90° C., the lower one under B by heating to 45° C., and the lower one under C by exposure to ultra-violet radiation. By comparing the lower organisms under B and C it may be seen that there is no difference in the appearance of these two organisms, both being slightly more opaque than the living organisms. The lower organism under B was killed by the coagulation of its protoplasm by heat, and since there is no difference in the appearance between this one and the lower organism under C, which was killed by exposure to ultra-violet radiation, it would seem to be fair to assume that the latter was killed by the coagulation of its protoplasm by the radiation. By comparing the lower organism under A with that under B it may be seen that the lower one under A is very much more opaque than the lower one under B. This greater opacity is explained by the fact that proteins are more firmly coagulated at a temperature of 90° C. than at a temperature of 45° C. It will be noticed also that the lower organisms under B and C which were killed by heating to 45° C. and by exposure to ultra-violet radiation respectively had begun to disintegrate, while the lower one under A had not begun to do so because of the firmer coagulation of the protein of this organism heated to the higher temperature.

Henri (5), Hertel (6), and others observed that when protozoa were exposed to ultra-violet radiation the body became swollen, drops of water appeared on the surface, and the organisms finally disintegrated, but they did not observe any coagulation produced by the radiation. The failure of these observers to obtain observable coagulation in the organisms was due to the fact that the radiation to which they exposed the organisms was not of sufficient intensity to coagulate firmly the protoplasm. If paramecia are heated to 40° C. they are killed after a time, but very little indication of coagulation is produced as is indicated by the fact that there is very little decrease in the transparency of the organisms thus killed. By increasing the temperature, however, at which the organisms are killed, the protoplasm becomes firmer

and the animals more opaque. Similarly by killing the organisms by exposure to ultra-violet radiation of low intensity, a very inconspicuous amount of the coagulation is produced, and hence there is very little change in the transparency of the organisms. If the intensity of the radiation is increased, however, the coagulation of the protoplasm becomes more marked with the resulting increase in the opacity of the animals.

*Action of the Different Wave Lengths in the Ultra-Violet Region of the Spectrum.* An extract of twelve pig lenses was made with 25cc of 0.1% calcium chloride solution and filtered through a coarse-grained filter. Five cc of this fairly clear filtrate were introduced into a circular quartz cell made of two quartz disks having between them a ring of hard rubber 0.8mm thick with an inside diameter of 3.8cm. By means of a small quartz spectrograph the radiation from a quartz mercury-vapor burner operating at seventy volts was focused on the lens extract in the quartz cell. The slit of the spectrograph was 1mm wide and the quartz burner was 3cm from the slit.

Figure IV. (1) is a photograph of the spectrum that was focused on the lens extract. (2) is a photograph of the lens extract after the spectrum had been focused on it for thirty hours. The coagulated line of lens protein, where the spectral line of wave length  $254\mu$  was focused, appeared after fifty minutes' exposure; that where the spectral line of wave length  $265\mu$  was focused, appeared after sixty-five minutes' exposure. The remaining lines of coagulated lens extract appeared after about two hundred minutes' exposure. (3) is a photograph of the spectrum made on a photographic plate with half the slit of the spectrograph covered with the cornea of a rabbit. It may be seen that the cornea transmits wave lengths as short as  $297\mu$  and  $302\mu$  and in (2) it may be seen that these wave lengths are effective in changing the protein of the lens so that calcium salts can combine with it to form a coagulum. (4) is a photograph of the spectrum made through a layer of the lens extract 1mm thick. It may be seen that the extract absorbs all wave lengths shorter than  $313\mu$  and in (2) it may be seen that all of these absorbed short wave-lengths are effective in coagulating the protein of the lens extracted with

0.1% calcium chloride solution. (5) is a photograph of the spectrum through a layer of aqueous humor 1mm thick.

In another experiment egg-white was introduced into the same quartz cell that had been used with the lens extract. (8) is a photograph of the spectrum of the quartz mercury-vapor burner. (7) is a photograph of the region of the quartz cell containing the egg-white where the spectrum had been focused for thirty hours. (6) is a photograph of the spectrum through a layer of egg-white 1mm thick. It may be seen that the egg-white absorbs wave lengths shorter than  $302\mu$  and that all these absorbed wave lengths are effective in coagulating the proteins of the egg-white. The periods of time required for the formation of the lines of coagulated egg-white where the spectral lines were focused are indicated in Figure IV., under "time for coagulation." It may be seen that the time required for the coagulation of egg-white by the different spectral lines is the same as that required for the coagulation of the modified lens protein by the corresponding lines.

*The Production of Anterior Eye Trouble in Living Animals by Means of Ultra-Violet Radiation.* One batch of six frogs was kept living partially immersed in 0.2% sodium silicate, another batch in 0.8% calcium chloride, another in 1.0% dextrose, another in tap water for fifteen days. At the end of this time one eye of each frog was exposed one hour each day for five successive days to the radiation from a quartz mercury burner operating at 140 volts, 3.3 amperes, and 2400 cp. at a distance of 20cm. Photographs of the frogs were made fifteen days after the exposures. Figure V., frog (1) had been living partially immersed in tap water previous to the exposures. Frog (2) had been living partially immersed in 0.2% solution of sodium silicate previous to the exposures. It may be seen that the eyelid of the frog living in the solution of sodium silicate had been converted by the radiation into an opaque mass, while that of the frog living in tap water was very little injured. The solution of calcium chloride had the same effect as the solution of silicate. The dextrose was effective but not so much as either of the other solutions. The conclusion may be drawn that salts such as are found to be greatly increased in human cataractous lenses increase the



effectiveness of ultra-violet radiation in producing anterior eye trouble.

It is a common experience that the skin sunburns more easily and quickly when it is wet than when it is dry. It is probable that if the skin is dry when it is exposed to the sunlight, the ultra-violet radiation in the sunlight changes the protein of the cells of the skin in such a way that salts such as those of calcium in the lymph bathing the cells can combine with it to form a coagulum. If the skin is wet with ordinary fresh or salt water the salts in the water facilitate the process by combining with the proteins of the cells modified by the ultra-violet radiation.

*The Production of Cataract in Living Animals by Means of Ultra-Violet Radiation.* An attempt was made to increase in the fluids of the body of living animals and hence in the eye media the salts found to be greatly increased in human cataractous lenses with the hope that on exposure of the eyes of these animals to ultra-violet radiation cataract would develop. Many observers have demonstrated that it is impossible to produce an opacity of the lens or cataract in the normal living animal by exposure of its eyes to ultra-violet radiation. Moreover, it is shown in this paper that it is not possible to produce an opacity even in excised pig lenses by exposing them directly to the radiation from a quartz mercury burner for very long periods of time. Fish were chosen for the experiment because they could be kept alive in the solutions of salts desired. One batch of goldfish was kept in 0.8% calcium chloride solution, another in 0.8% calcium nitrate solution, another in 1% dextrose, another in 0.1% sodium silicate for ten days. At the end of this time each fish in its turn was introduced into a small box with a quartz window in one side. In practice four of these boxes were used so that one eye of each of the four fish was exposed simultaneously. Fresh cool water was kept circulating through these boxes during the exposures. The boxes containing the fish were adjusted so that the quartz windows were 15cm from the quartz mercury-vapor burner operating at 140 volts, 3.3 amperes, and 2400 cp. In this manner one eye of each fish was exposed to the radiation. Each exposure was of six hours' duration. After the exposure, the batches

of fish were replaced in the solutions from which they had been removed. For comparison, the eyes of fish living in tap water were exposed in the same manner and for a similar length of time as those living in the salt solutions. As a rule a slight opacity in the cornea of the eye exposed appeared about fifteen hours after the first exposure. It was assumed that this opacity did not develop during the exposure because there was not sufficient salt present in the cells of the cornea to combine with the protein modified by the radiation to form a coagulum. However, at the end of twelve or fifteen hours a sufficient quantity of salt had collected, owing to the diffusion of the salt from the blood stream into the cornea, and had combined with the modified protein to precipitate it and hence an opacity of the cornea was produced. This assumption would seem to explain the so-called "latent period," *i. e.*, the time elapsing between the exposure of one's eye to ultra-violet radiation and the time when the painful effect is felt. In most of the fish a slight clouding appeared about two days after the first exposure in the lens of the eye exposed.

Ten days after the first exposure the eyes of the fish were exposed again for another six-hour period. At the time of this second exposure as a rule an opacity of the cornea and of the lens of the fish living in the salt solutions had increased while the opacity of the cornea of the fish living in tap water had practically cleared up. Several hours after the second exposure as a rule the opacity of the lens and of the cornea of the fish living in the salt solutions became more marked. An opacity of the cornea of the fish living in tap water developed also but this was slight and cleared up in a few days while that of the fish living in the salt solutions increased.

After the second exposure no prescribed rule as to time for the third exposure can be laid down. In order to clear up the opacity of the cornea of the fish in the different salt solutions it was necessary to transfer them to tap water. As a rule the opacity of the cornea cleared up in a few days while the lens remained opaque. By nursing, by exposing to ultra-violet radiation, by transferring back and forth from salt solution to tap water, it was possible to obtain fish in the condition indicated in Figure VI. Fish (1) had been living in tap water for thirty days and had been exposed to ultra-violet

radiation for two six-hour periods or twelve hours. Fish (2) had been living in 0.1% sodium silicate for twenty-eight days and had been exposed to ultra-violet radiation for two six-hour periods or twelve hours. Fish (3) had been living in 0.1% sodium silicate for forty-two days and had been exposed to ultra-violet radiation for four six-hour periods or twenty-four hours.

It may be seen that the lens of fish (3) living in the silicate solution and exposed to ultra-violet radiation for twenty-four hours had become perfectly opaque; that of fish (2) living in the same solution but exposed to ultra-violet radiation for twelve hours had become partially opaque, while the lens of fish (1) living in tap water and exposed to ultra-violet radiation for twelve hours was practically clear.

In Figure VII. are shown the photographs of the spectra of the small quartz mercury-vapor burner operating at 70 volts and that of a large quartz mercury-vapor burner operating at 140 volts. Photographs showing the effects produced by focusing these spectra on egg-white and on lens protein are also given in order to show that the most effective regions of the two spectra in precipitating protein are different. (1) is the photograph of the spectrum of the small quartz mercury-vapor burner made on a photographic plate, (9) is a photograph of the spectrum of the large quartz mercury-vapor burner made on a photographic plate also. By comparing these two spectrograms it may be seen that the spectral lines in the extreme ultra-violet region of the spectrum are much more intense in (1) than in (9). (2) is a photograph of the cell containing egg-white on which the spectrum of the small quartz burner had been focused for thirty hours. (8) is a photograph of the quartz cell containing egg-white on which the spectrum of the large burner had been focused for the same length of time. By comparing (2) and (8) it may be seen that the egg-white was coagulated in (2) where the spectral lines of wave lengths  $302\mu$  to  $249\mu$  were focused, while in (8) the egg-white was coagulated only where the spectral lines of wave lengths  $302\mu$  and  $297\mu$  were focused. (3) is a photograph of the quartz cell containing lens protein extracted with 0.1% calcium chloride on which the spectrum of the small burner had been focused for thirty hours. By

comparing (3) with (2) and (7) with (8) it may be seen that the same region of the spectrum of the small quartz mercury-vapor burner effective in coagulating the protein of the egg-white is also effective in coagulating the lens protein extracted with 0.1% calcium chloride solution. The same statement may be made regarding the effective region of the large quartz mercury-vapor burner in (7) and (8). (4) is a photograph of the spectrum of the large quartz mercury-vapor burner made through the cornea of the rabbit, (5) made through glass 1mm thick, (6) through glass 5mm thick. It may be seen from (4) that the cornea transmits wave lengths as short as  $297\mu$  and  $302\mu$  and from (7) that these are the wave lengths of the spectrum of the large quartz mercury-vapor burner most effective in bringing about the coagulation of the protein of the lens. The large quartz mercury-vapor burner was the one used in the experiments described in which cataract was produced in fish living in salt solutions.

Many observers have attributed their failure to produce cataract in living animals to the protective action of the cornea. I think it will be clear from the experiments reported in this paper that their failure was not due to the protective action of the cornea but to the fact that there were not present in the lens of the eyes of the animals exposed sufficient quantities of salts such as those of calcium to combine with the protein of the lens modified by the ultra-violet radiation, to precipitate it.

#### CONCLUSIONS.

1. Sodium and potassium salts in sufficient concentration act specifically on the nucleus of the lens, producing nuclear opacity, while calcium salts act specifically on the cortex of the lens, producing cortical opacity.

2. The short wave-lengths of the spectrum produce a molecular rearrangement in the protoplasm of the cells of the crystalline lens, so that inorganic salts such as are found to be greatly increased in human cataractous lenses can combine with the protoplasm to precipitate it, and hence produce an opacity. That the change produced in protein by exposure to ultra-violet radiation is molecular in character and not atomic

was shown by the fact that the exposure did not affect the conductivity of the protein.

3. Since ultra-violet radiation produces a molecular rearrangement in the living material of the lens so that weak solutions of inorganic salts can combine with it to form a precipitate, and since sodium salts are specific for the modified protein of the nucleus and calcium salts for that of the cortex, the assumption is made that of the two general types of cataract, nuclear and cortical, sodium salts function in the production of nuclear and calcium salts in the production of cortical cataract.

4. The lens protein is not only rendered more sensitive to the action of certain inorganic salts by exposure to ultra-violet radiation, but is also rendered more sensitive to the action of certain dyes, as is shown by the fact that lens protein exposed to ultra-violet radiation takes certain dyes more readily than the unexposed protein.

5. That ultra-violet radiation kills living cells by coagulating their protein may be seen by direct observation through the microscope during an intense exposure of unicellular organisms such as paramecia.

6. The effective region of the spectrum in changing the living material of the cell or protoplasm lies between  $254\mu\mu$  and  $302\mu\mu$ . The most effective region is around  $254\mu\mu$  for the small quartz mercury burner used, and around  $302\mu\mu$  for the large burner.

7. An opacity of the lens, or cataract, can be produced in fish living in solutions of those salts found to be greatly increased in human cataractous lenses by exposing the eye of the fish to the radiation from a quartz mercury-vapor burner. This cannot be done by exposing the eyes of fish living in tap water, which contains only very small quantities of these salts.

8. Abnormal quantities of the salts of calcium and sodium silicate in the cells of the eyelid and of the cornea increase the effectiveness of ultra-violet radiation in producing trouble in these structures. Abnormal quantities of calcium salts on the skin presumably increase the effectiveness of the short wavelengths in sunlight in producing sunburn.

9. In looking for the cause of cataract it would seem that

at least two factors should be considered, the one a modification of the protein of the lens by ultra-violet radiation, and the other certain inorganic salts by which the modified protein can be precipitated. According to this hypothesis, the prevalence of cataract among people living in the tropics could be accounted for by the increase in the radiant energy factor modifying the lens protein so that an excess of salts, such as silicates in case of people in India, would combine with the protein to precipitate it and produce an opacity of the lens or cataract. The prevalence of cataract among glass blowers is also accounted for by the excess of the radiant energy factor, the assumption being that glass blowers who develop cataract have a more or less disturbed condition of nutrition expressing itself in an increase in sugar in case of diabetics, calcium salts, or some other substance, which can combine with the lens protein made sensitive by the action of the short wave-lengths. The prevalence of cataract among diabetics is accounted for by the increase, not in the radiant energy factor, but in the chemical factor, specifically dextrose, acetone,  $\beta$ -oxybutyric acid, etc.

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## SALVARSAN AND OTHER REMEDIES IN OPHTHALMIC PRACTICE.

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POSSIBLY in no region of medical science is it more difficult to estimate results accurately than in the very important domain of therapeutics. There are often so many factors that are quite unascertainable complicating the problem that it is difficult to estimate how far any drug is of use. Every now and again some remedy is loudly vaunted as being invaluable in certain forms of disease, but after a few years it passes out of notice and is no longer thought suitable for the very conditions for which it was regarded as a specific on its first introduction. That there has been material progress in therapeutic work must be generally admitted, but what real and lasting progress has been made in recent years has been almost entirely on the biological side, and probably we are only at the beginning of still greater advances in the same direction. When a remedy is at one time extolled as being of very special use under certain conditions, and a few years afterwards is rarely if ever mentioned, it suggests that the original introduction of that medicine was made without a thorough and impartial test. Possibly even in some cases it is boomed by manufacturers who have a financial interest in putting it on the market. One or two examples will illustrate our meaning. Some years ago certain organic preparations of silver were supposed to supersede everything else in the treatment of various forms of conjunctivitis. Three of the best known were protargol, argyrol, and collargol. The first was largely brought into notice by a book written by Dr. Darier of Paris and translated for the English public by Mr. Sydney Stephenson.

A more disappointing book we never read. It is absolutely destitute of any scientific information on the subject. It is a piece of mere empiricism and tolerably crude at that. How these remedies came to be popular and sold in the way they were, as germicides with important actions, is a mystery, for if there is one thing certain, it is that their germicidal actions are practically negligible. Many experiments have been made with strong solutions of these drugs. Cultures have been immersed in solutions of them for hours together and then sub-cultures have easily been obtained. It is difficult to believe that anybody can still hold that a few drops of a solution of any of these drugs introduced into the conjunctival sac for a few seconds can have any influence whatsoever on the flora of the conjunctiva. Undoubtedly they give better results than strong solutions of nitrate of silver, such as were at one time frequently used, for the simple reason that these preparations do no harm, which nitrate of silver unquestionably did. Looking back on the results of the treatment of gonorrheal ophthalmia with strong solutions of nitrate of silver, we cannot but think the result was in almost every case detrimental. The conjunctival sac was never sterilized although the protecting epithelium of the conjunctival membrane and sometimes that of the cornea were seriously damaged. The organic preparations already mentioned do no harm, nitrate of silver does, and hence these preparations appeared to the authors whom we have indicated as intrinsically of high value.

It has now been known for a considerable time that these drugs in the treatment of conjunctivitis are inert, and yet patients turn up in large numbers at our dispensaries and in our private clinics who produce letters from their medical advisers to say that they have been using protargol or argyrol for some weeks or months but without effect. Nobody who knows the facts of the case would expect anything else. Many years ago, indeed shortly after commencing the study of ophthalmology, I came to the conclusion that a conjunctivitis was the expression of one of two things, namely, either an uncorrected error of refraction or a microörganic infection. In the former case it is a question of measuring the error of refraction and correcting it accurately. In the latter it



is a matter of removing and not of killing the organism or organisms.

Another instance of a method of treatment which has been largely boomed was Röntgen ray and radium treatment for cancer. I do not suppose there is a single instance on record in which a case of cancer has been cured by these means, any more than there is on record a case of cancer which was cured by the Count Mattei blue or white electricities. It is possible, perhaps, that the employment of these remedies may give some relief in cases of inoperable cancer and may make the sufferer a little more comfortable, but evidence is totally lacking that treatment by radium or treatment by the Röntgen rays has ever cured cancer. Both of these remedies are becoming more and more placed on the shelf for that malady.

A third typical example is the difference between antiseptic and aseptic treatment. In the old days gauze dressings for wounds used to be thoroughly impregnated with carbolic acid. I myself have examined some of the discharges contained on these dressings and found them to be teeming with micro-organic life. Lister's application of Pasteur's great discovery, we venture to think, was the happiest that has ever been made in the science of medicine, but he did not realize that the means which he proposed for the destruction of germ life were not adequate for the purpose. It is no more wonderful that he did not see the whole of the ground which his work opened than that Isaac Newton saw the fallacies in the corpuscular theory of light, yet, as in the case of protargol, the old idea still persists.

We still find ophthalmic text-books figuring an undine and expatiating in the use of that instrument for applying boric solution as an antiseptic. That a special form of instrument should thus be widely advertised may be of use to the persons who have these appliances for sale. From our point of view it is probably the worst contrivance. You cannot kill germs, as far as we know, by anything which you can apply to the conjunctiva without destroying the membrane itself. You may remove the germs and thereby save the situation, but you will require a thoroughly good douche with a fall of at least eighteen inches and not less than a pint at a time to be used, and of all the apparatus for carrying out such a line of treatment

probably the undine is the least useful. When we find an instrument of that sort recommended, we cannot help suspecting that the author who advises it is still under the impression that the ingredients of the lotion are the important thing and not its method of application. With very few exceptions, the only useful element in any lotion is water which, if not to be painful, must be made isotonic with something or other, preferably with sodium chloride. We cannot help thinking that a man who prescribes boric lotion to be used in an undine has done very little observational work in conjunctival bacteriology.

There is, as is well known, the exceptional case of the use of zinc sulphate apparently freeing the conjunctival sac of the Morax parasite. No doubt similar instances of germicidal effects may be discovered, but apart from this, so far as we know, there is no other instance of an ordinary drug causing an infective microorganism to disappear so long as the remedy is applied only in such strengths as will do no harm to the conjunctival membrane. No one can foretell what the future may have in store, but that is pretty much how the matter stands at present.

But a very important question now arises. Of recent years the same country which gave us protargol and argyrol has given us a new remedy for syphilis for which the strongest claims have been made in influential quarters. It is said to be infinitely better than anything that has yet been used. Men specially trained are to be appointed by the State for its administration and no doubt a large section of the British public who are the victims of syphilis will have salvarsan or its substitutes administered to them.

The circumstance that certain practitioners are to be appointed for its administration points clearly to one thing, and that is that its administration is by no means free of danger, and indeed there is a very fair death-roll connected with the use of this drug already. The vital question thus comes to be: Is this treatment so overpoweringly superior to any other that we are justified in running the extra risk? Is salvarsan or neo-salvarsan so immeasurably superior to mercury that we are justified in running a moderate percentage of risk? If it were proved that these new drugs were an infallible remedy for

this terrible disease I would answer that question in the affirmative.

That however is very far from being the case; and in the state of doubt as regards this line of treatment in which I find myself I have ventured to record some cases to which I would, with all respect, call the attention of syphilologists. I am not one myself but have every confidence that those who are working in this particular department will ultimately get at the facts of the case. I see no more syphilis than is usually seen by ophthalmic surgeons in hospital and private practice.

To begin with, I found that salvarsan was a failure in interstitial keratitis. So far as that disease is concerned, mercury has given very much better results than these newer drugs. I believe that my experience is not singular in this respect. Other practitioners have also reported against their use in this particular disease. The explanation of this failure given by the advocates of this new line of treatment is that the cornea is a special tissue and that the drug does not reach it.

Now one thing is certain and it is that although in health there are no blood-vessels in the cornea still it contains a large number of lymphatics. The fluid which these contain is no doubt derived from the blood stream; why then should the drug not find its way into the tissue? I am at a loss to find an answer.

And here perhaps I may be allowed a short although, as it seems to me, an important digression. Recently I read a statement which in my opinion underestimated the gravity of interstitial keratitis. It was to the effect that the prognosis of this disease is favorable and that the cornea clears completely. Now my experience is that although in the main the prognosis is not bad yet I have never seen a cornea which was not to some extent damaged, and that permanently, by this form of keratitis. In some cases the injury may be slight, in others it is severe and materially interferes with vision.

My present purpose, however, is to call attention to five cases in which the organ of vision was involved by syphilitic disease otherwise than by keratitis and in all of which salvarsan or neo-salvarsan had been used. I shall make almost no comments but for the most part will simply record them as they occurred.

The first case is that of an officer who some six years ago consulted me for an acute iritis. He was put on mercury and atropine in the usual manner and the condition began steadily to improve. He had heard of the new treatment of syphilis and was anxious to try it as a permanent cure. I directed him to avail himself of the services of a colleague who is thoroughly authoritative in this line of practice. That gentleman was given a perfectly free hand and gave him what injections he thought proper. Thereafter a Wassermann reaction was taken and he was pronounced to be permanently cured of his disease, and all treatment was stopped. Ten months afterwards he re-appeared in my room, this time complaining of severe pains in his legs. I sent him to the same practitioner, who found that the Wassermann was now positive. He removed to another station and I cannot tell what became of him. I could not discover any definite ataxic symptoms, but at the back of my mind there was the idea that the pains in the legs might be the beginning of locomotor ataxy.

The second case which occurred about the same time is also very important. A patient came complaining of defective vision in the left eye. On examination I found it to be due to a slight optic neuritis. He admitted quite freely that he had syphilis. Like the patient whose case has just been narrated, he also had heard of the new treatment as a perfect and permanent cure of syphilis and was anxious to try it. I at once gave my consent and directed him to the practitioner who had administered the drug to the patient already mentioned. The injections were duly given and after a Wassermann reaction was found to be negative he was pronounced cured. Within six months he returned to see me and I found him suffering from an acute iritis which at once yielded to treatment with mercury. There had been no chance of a re-infection.

The third case is of special importance, for it suggests even more strongly than the first that instead of being beneficial neo-salvarsan had been injurious. The patient was a lad who, many years before the introduction of salvarsan treatment in any of its forms, acquired syphilis. When I saw him he had all the signs and symptoms of well-marked paralysis of the external rectus of the *right* eye. He was at once put upon mercury by the mouth and by inunctions and made a

thoroughly good recovery. A good number of years afterwards he heard of neo-salvarsan as a remedy which would entirely remove syphilis from his system. Naturally he consulted a practitioner who is justly much esteemed in this line of practice and the drug was duly administered to him. Two years afterwards he came to me complaining of complete blindness of the *left* eye. I found advanced optic nerve atrophy in the affected organ. He told me that he had not observed anything wrong with the eye before the administration of the drug, but that shortly thereafter he found the vision affected and that it had become gradually worse till complete blindness supervened. The administration of the drug may have had nothing to do with the onset of the atrophy, but in view of the facts which we know about soamin, which was previously supposed to be a perfect and safe cure for syphilis, the onset of the atrophy could not but raise suspicions.

The fourth case is one in which the patient had no fewer than eight injections. The first four were given in July and August, 1916, and the last in March and April, 1917. In August, 1917, he came to me complaining of diplopia, which I found to be due to complete paralysis of the internal rectus of the right eye. The movements upwards and downwards were also slightly restricted and there was slight dilatation of the right pupil. The visual acuteness of the eye was  $\frac{6}{12}$ . He had also all the symptoms of spinal sclerosis. It was quite evident that in this case the drug had not so relieved him of the protozoön as to prevent nerves hitherto unattacked becoming affected.

The first, third, and fourth cases help to strengthen the view that salvarsan, containing as it does arsenic, may be specially harmful to nerve tissue.

The fifth and last case also would seem to show that this method of treatment is not of permanent value. The patient received five injections in Sydney about the month of February, 1916. A Wassermann was done and he was pronounced to be perfectly free of the disease. In July of this year he was admitted to the Fourth Scottish General Hospital with an acute choroido-iritis of the left eye, and so authoritative an observer as Professor Muir pronounced the Wassermann to be again positive.

My experience of syphilis is small. One swallow does not

make summer, and five even such striking cases as the above do not entitle one to do more than hold his opinion in reserve, but that I certainly am doing. I would suggest, however, that all cases in which the drug has been used should be registered and followed up for some years. If the drug is more efficacious than mercury and more lasting in its results and does not cause any ulterior damage, then it may be justifiable to run a certain risk, but if not, then it should not be used. I have formed no dogmatic opinion and am prepared to consider carefully any well ascertained facts which may be stated by those who have a large practice amongst the unfortunate victims of this malady.

## FIVE YEARS' EXPERIENCE WITH IRIDOTASIS.<sup>1</sup>

BY DR. DAVID HARROWER, WORCESTER, MASS.

*(With two figures in text.)*

IN 1911 Borthen gave his experience with the operation of iridotasis as devised by him in ninety-seven cases. The results obtained by him were so remarkable that I was led to try the operation in 1911. I reported two cases before this Society in 1912, and several more in 1913. All these cases were done for chronic glaucoma, or for the relief of pain in blind eyes. Of these, two were for the relief of pain, and both were successful. In all I have done twenty-three, so far, with improvement in eyes where there was vision. The field has increased, the tension has diminished, and the patient has been made comfortable at once.

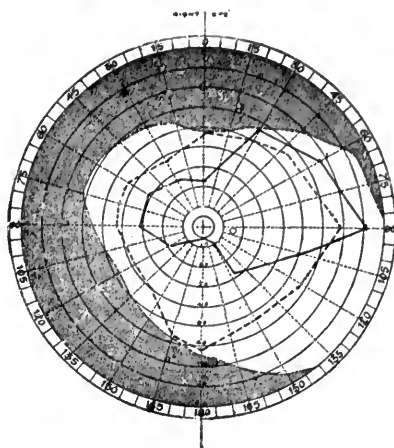
I have so far followed the directions of Borthen as near as possible. I instil a drop of 1% atropin fifteen minutes before I intend to operate. I then follow with a 4% solution of cocaine every few minutes for ten minutes before I begin my operation. For the sake of those who may not have read the method, I will briefly describe it.

After the cocaine has been used ten minutes, I grasp the conjunctiva about 10mm back from the cornea, and make a cut about 10-12mm parallel with the corneal line. It is important that the whole subconjunctival tissue should be included right down to the sclera. This whole tissue should be now separated from the sclera down to the sclero-corneal junction, being very careful not to buttonhole, as that would lead to infection. If the conjunctiva is punctured a new field should be used. I use the inverted scissors in dissecting the

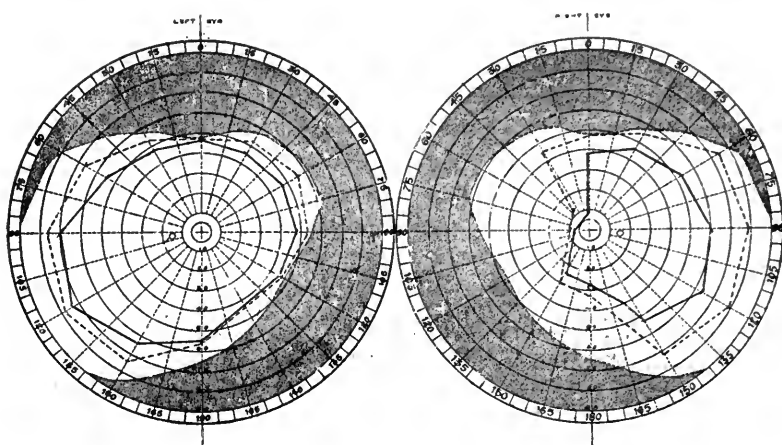
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<sup>1</sup> Read at meeting of American Ophthalmological Society, 1917.

conjunctiva down to the corneal margin. If the patient is quiet, I simply fix the eye by grasping the free edge of the



CASE 1.—Solid line shows field January 26th; dotted line, field April 4th



CASE 2.—Right eye, solid line shows field, March 23d; dotted line, field May 4th. Left eye, solid line shows field April 17th; dotted line, field May 4th.

conjunctiva with the fixation forceps. Asking the patient to look down, I make the incision just behind the corneal margin about 4mm wide. Then with a pair of iris forceps introduced



through the opening just made, I grasp the pupillary margin of the iris, withdraw the iris into the opening in the sclera, smooth back the conjunctiva into place, and the operation is over. If the patient is the least unquiet, I have an assistant fix the eye for me, but so far I have only twice had to use one. I have had no complication, except as in Case 2 and in Case 5 in my report to the Society in 1913, both of which recovered nicely.

I think it very important that the whole conjunctival tissue down to the sclera be included in the operation, as it seems to help the filtration. Borthen lays great stress on the stretching of the iris. He says: "I am convinced that the operation itself supplies a number of important factors which have to do with the structure and position of the iris. Inspection of these eyes after operation shows a very marked variation in the form of the pupil from that produced by iridectomy. It is oval or pyriform with the point toward the incarceration. The radial arrangement of the iris fibers, too, is entirely changed, so that instead of converging toward the central pupil, they are drawn out in the direction of the prolapse. The crypts appear longer, and the contraction furrows are smoothed out. This traction on all parts of the iris diaphragm is marked and may be so excessive as to draw the pupil away from the center. I believe that this traction itself indirectly affects tension much in the same way as, but more actively than, eserine and active accommodation. I am convinced that extraocular filtration is only one, and possibly a minor, factor in the reduction of tension, and the increased drawing through Fontana's spaces in direct consequence of the stretching of the iris plays an important rôle." Personally, I think that the infiltration bleb properly protected with the largest amount of conjunctival tissue is the important part.

Since I made my report of 1913, several reports of cases have appeared. Dr. Dunbar Roy, of Atlanta, Ga., has reported several cases. Dr. Stieren, of Pittsburg, Pa., reported eight cases with good results. Two of my colleagues in Worcester have done this operation. One has done eight cases all with good results; one three cases all with good results.

I have not tried Borthen's recent modification, as his original operation has done so well. I have done twenty-three

operations in all. Every one of them so far has been successful. On May 15th I saw the first two cases that I operated on, and they both had held the vision that they obtained after the operation, and also their fields and tension.

I report the following cases that are typical of all I have so far operated on:

CASE 1.—Mr. A. P., aged 65, March 8, 1916, came to me complaining of severe headaches and failing vision. He had been under treatment for his failing vision for nearly a year. O. D.  $\frac{1\frac{5}{8}}{10}$  + 1 D. cyl. ax.  $70^\circ = \frac{1\frac{5}{8}}{8}$ , T. 30mm. O. S.  $\frac{1\frac{5}{8}}{4}$ ? + 1 D. sph. =  $\frac{1\frac{5}{8}}{2}$ , T. 57mm. The nerve was excavated, especially in the right eye. He was put on 1% pilocarpin.

April 14. O. D.  $\frac{1\frac{5}{8}}{5}$  with correcting glass T. 30mm; O. S.  $\frac{1\frac{5}{8}}{3}$  with correcting glass T. 40mm.

Sept. 5. O. D.  $\frac{1\frac{5}{8}}{5}$  with correcting glass T. 30mm; O. S.  $\frac{1\frac{5}{8}}{2}$  with correcting glass T. 40mm.

Jan. 26, 1917. O. D.  $\frac{1\frac{5}{8}}{5}$  with correcting glass T. 60mm; O. S.  $\frac{1\frac{5}{8}}{2}$  with correcting glass T. 35mm.

Feb. 3. O. D.  $\frac{1\frac{5}{8}}{4}$  with correcting glass T. 40mm; O. S.  $\frac{1\frac{5}{8}}{2}$  with correcting glass T. 30mm.

Feb. 5. As the field was contracting in the right eye I did iridotaxis.

Feb. 21. O. D.  $\frac{1\frac{5}{8}}{4}$  with correcting glass T. 22mm; O. S.  $\frac{1\frac{5}{8}}{2}$  with correcting glass T. 25mm.

May 17. O. D.  $\frac{1\frac{5}{8}}{4}$  with correcting glass T. 22mm; O. S.  $\frac{1\frac{5}{8}}{2}$  with correcting glass T. 25mm.

CASE 2.—Mr. J. H. F., aged 61, March 19, 1917, referred to me by Dr. John Cahill. Seen by him Nov. 11, 1916, complaining of failing vision. Vision at that time was with correcting glasses O. D.  $\frac{1\frac{5}{8}}{20}$ , O. S.  $\frac{1\frac{5}{8}}{5}$ . Was given pilocarpin, but as no improvement followed was referred to me.

March 23. Field in left eye as shown.

March 26. I did iridotaxis on right eye.

April 3. O. D. T. 25mm. O. S. T. 25mm.

April 17. O. D.  $\frac{1\frac{5}{8}}{10}$ ?, O. S. + .50 D + 1.50 D. cyl. ax.  $180^\circ = \frac{1\frac{5}{8}}{6}$ .

April 21. Field of left eye more contracted. T. 34mm.

April 23. Did iridotaxis on left eye.

May 4. O. D. + .75 D.  $\frac{1\frac{5}{8}}{10}$ . T. 25mm. O. S. + 1.50 D. cyl.  $180^\circ = \frac{1\frac{5}{8}}{6}$ . T. 25mm.

May 12. Vision in left eye is still  $\frac{1\frac{5}{8}}{6}$ .

May 18. Vision in left eye is  $\frac{1\frac{5}{8}}{7}$ . T. 30mm. Not a good bleb.

May 21. O. S. vision is  $\frac{1}{50}$ . Still no good bleb. T. 25mm.

May 25. O. S. vision is  $\frac{1}{50}$  with glass. Small bleb beginning to show. T. 24mm.

CASE 3.—Operated in Boston before the American Surgical Society: "Case 31. No. 4254—Age 67, M. Simple glaucoma, left, tension 7mm. Operation Oct. 27th, cocaine. Dr. Harrower. Iridotaxis. No complications. Discharged Oct. 31st, improved. End-results: eye white and quiet; tension 10mm; filtration scar." A report six months after the operation showed improvement.

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## LATE INFECTION WITH ENUCLEATION FOLLOWING AN OPERATION OF IRIDOTASIS FOR CHRONIC GLAUCOMA.<sup>1</sup>

BY DR. DUNBAR ROY, ATLANTA, GA.

(With one illustration on Text-Plate IV.)

THE question of late infection following the trephine operation for glaucoma has been quite prominent during the last few years and a number of such cases have been reported by different observers. While the number of operators and the number of such operations have been far in excess of those performed according to the technic of iridotasis, yet up to the present there has been no case reported of late infection following iridotasis, making the present report the first case on record.

This case has already been mentioned by me in the series of nine cases operated upon by the technic of iridotasis and which were reported in the *Ophthalmic Record* for last year.

The history is as follows: Mrs. W. T. R., age 49, consulted the writer on March 7, 1912. According to the patient, she had always had some degree of weakness with her eyes, but about six weeks previous she began to have some dimness of vision, slight pain, and halos around the lights at night. The right eye is especially weak and irritable.

R. Eye Vision =  $\frac{20}{160}$  and with myopic correction  $\frac{20}{40}$ .

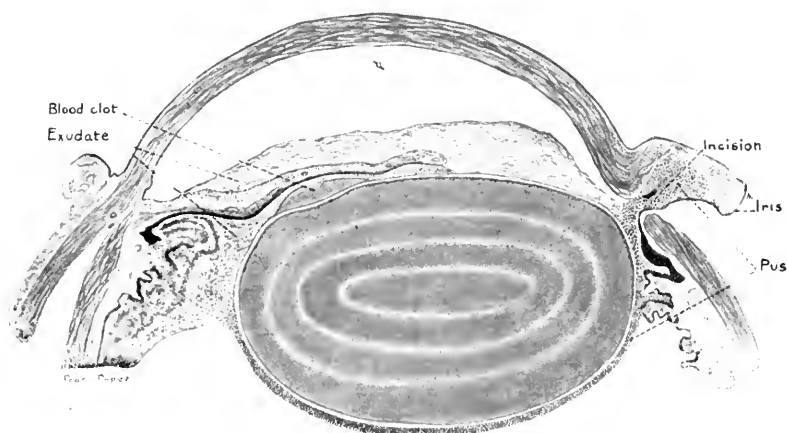
L. Eye Vision =  $\frac{20}{20}$  and with myopic correction  $\frac{20}{40}$ .

The right eye showed some increase in the intraocular tension. Pupil slightly dilated, iris bombe. Media clear and no cupping of the disk. Slight congestion and neuralgic pains. The left eye showed normal tension, fundus clear, and no cupping. Correction with glasses were pre-

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<sup>1</sup> Read at meeting of the American Ophthalmological Society, 1917.

ILLUSTRATING DR. ROY'S REPORT OF A CASE OF "LATE INFECTION WITH  
ENUCLEATION FOLLOWING AN OPERATION OF IRIDOTOMY FOR  
CHRONIC GLAUCOMA."





scribed and a weak solution of eserine ordered to be used three times daily in right eye. Letters from the patient indicated that she was more comfortable and was feeling well satisfied with her condition. On July 1st, same year, patient wrote that her vision in the right eye had become worse and that there was much more pain. On July 7th, she came to the city and the writer operated the same day on the right eye under cocaine anæsthesia by the method of iridotaxis. No difficulty was experienced and all symptoms relieved. No suture was made in the conjunctival flap, which the writer now believes was a serious mistake, this being the second case he had operated upon by this method. Since then sutures have been used in every case. The patient returned home on the fifth day and experienced no further trouble.

On July 24th, thirteen days after the operation, patient returned. The iris and conjunctival bleb seemed in good condition and vision had improved to  $\frac{20}{40}$  with correction and free from all discomfort. Tension was apparently normal.

The patient did not find it necessary to consult me until one year later. In June, 1913, the left eye was seized with the same acute glaucomatous symptoms as the right, which necessitated an immediate operation. This also was performed under local anæsthesia and the operation of iridotaxis was used. In this case a suture was placed in the conjunctival flap. The healing was uneventful and all symptoms relieved. Vision, two months after operation, with correction was  $\frac{20}{40}$ .

This patient remained perfectly comfortable and her vision in both eyes with correction was in no wise diminished for a period of almost five years.

On January 15, 1917, almost five years after the original operation, following a severe cold, the patient began to have pain and irritation in the right eye. This continued to grow worse. On January 22d, one week later, she again consulted me. The history showed that the patient had been thoroughly comfortable up to one week ago. There was absolutely no history of traumatism.

*Examination.*—Patient was suffering considerable pain. The lids upper and lower were swollen and oedematous, giving the patient almost the appearance of an orbital cellulitis. The bulbar conjunctiva was chemotic, the cornea hazy, and considerable protrusion of the iris. Pus was noted in the anterior chamber. The eye showed a typical picture of panophthalmitis.

The left eye was in no way affected and the previous operative results were all that could be desired. Under a general anæsthetic the right eye was immediately removed.

The only accident was the escape of the fluid contents of the eyeball through the original operative incision and the protruding iris. Healing was uneventful. The left eye still remains good. The collapsed eyeball was immediately sent to Dr. F. H. Verhoeff in Boston, who examined the specimen and sent the following report:

"The globe is partly collapsed owing to the large perforation in the sclera near the equator, made evidently during the removal of the eye, and as a consequence, the retina is partly separated. The bleb resulting from the iridotaxis is filled with pus and the iris tissue within it is disorganized therapy. The corneo-sclera shows a clean-cut patent opening into the bleb. The lens is not injured and is not incarcerated in the opening. On the opposite side the filtration angle is blocked by the root of the iris. The anterior chamber is filled chiefly with fibrinous coagulum. The pus in the bleb continues through the fistula into a mass of pus which completely fills the vitreous chamber. The ciliary body and retina are giving rise to an abundant exudation of pus cells. The choroid is markedly infiltrated with lymphoid cells, but shows practically no pus cells. The Gram stain shows an abundance of pneumococci in the pus throughout the vitreous chamber. On the ciliary body below there is a small mass of fibrous tissue pervaded by proliferated pigment epithelium, evidently the result of an old localized cyclitis. At the equator, the lamina vitreous of the choroid shows an unusually abundant formation of colloid excrescences. Diagnosis: Iridotaxis with late infection with pneumococci. Advanced purulent endophthalmitis."

*Remarks:* The fact that the case was one of late infection following the operation of iridotaxis will in no wise deter me from continuing to use this operation as the one of choice in acute and chronic glaucoma. There were two factors which contribute to this untimely accident and which would have to be considered in any operation for glaucoma.

In the first place, the right eye showed some slow choroidal changes at the original examination, which was also manifested by the myopic refraction. This evidently was progressing and produced a decided thinning of the tunics of the eyeball. Hence the inability of these structures to withstand this increased intraocular pressure and thus giving away at the point of operative incision. This was manifested by the large amount of protruded iris found at the time of enucleation.



In the second place, the failure to replace the conjunctival flap with a retention suture was a mistake, as the protruding iris should be well protected by the conjunctiva. In addition, too large an incision was made at the sclero-corneal margin, with the consequent too large a piece of iris pulled into this wound at the time of original operation. Since then the writer makes a very small incision which allows only a small piece of iris to be pulled up and therefore much more securely anchored in the wound.

## TENSION IN NORMAL EYES BEFORE AND AFTER TONSILLECTOMY.<sup>1</sup>

By ANDY M. CARR, A. B., M. D., CHICAGO,

Staff Resident in Ophthalmology, Cook County Hospital.

(From the service of Dr. E. V. L. Brown.)

THESE observations were made to determine what effect if any, the hemorrhage, anæsthetic, and shock of tonsillectomy have upon normal intraocular tension.

A number of cases of iritis with secondary glaucoma had shown a marked and permanent lowering of tension, along with striking improvement in the iritis, within twenty-four hours after the removal of abscessed tonsils. No tension change had occurred in the fellow eye.

The connection between the infected tonsils and the eye lesion seemed clear, yet it was thought best to make a control study of tension in normal eyes before and after tonsillectomy.

The material comprised one hundred cases of tonsillectomy in patients with no history of eye trouble. None but very controllable patients were used. Their ages ranged from 14 to 67 years. Pus was expressed from the tonsil in practically every case. About two thirds of the cases were operated under local anæsthesia, the remainder with ether. The tension was taken first a few hours before and then twenty-four hours after the operation. The Schiotz tonometer was used throughout and the eyes were anæsthetized with holocain.

From the accompanying table it may be seen that no variation greater than 2mm was noted in 95% of the cases. This

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<sup>1</sup> Read before the Chicago Ophthalmological Society, May 17, 1917. From the Research Laboratory of the Cook County Hospital, Chicago.

amount is of no significance, as all investigators agree that it is in the range of the normal error of observation and is due to the limitations of present-day tonometry. In no case was there a variation of 5mm in the readings made respectively before and after the tonsillectomy.

Two cases had secondary hemorrhage with a considerable degree of exsanguination. No difference was noted between local and general anæsthetic cases.

The average tension of the one hundred cases was  $17\frac{1}{4}$ mm; the lowest tension 10mm, the highest 26mm.

We therefore feel justified in concluding that tonsillectomy, even when accompanied by considerable hemorrhage, does not have any effect upon normal intraocular tension.

TABLE.

BEFORE TONSILLECTOMY.						AFTER TONSILLECTOMY.					
CASE	AGE	TENSION.		PUPILS.		TENSION.		PUPILS.		CHANGE.	
		Rt	Lt	Rt	Lt	Rt	Lt	Rt	Lt	RISE	FALL
1	19	13 $\frac{1}{4}$	15 $\frac{1}{4}$	4	4	13 $\frac{1}{4}$	16 $\frac{1}{2}$	4	4	1 $\frac{1}{4}$	
2	18	18	15 $\frac{1}{4}$	4	4	18	16 $\frac{1}{2}$	5	4	1 $\frac{1}{4}$	
3	17	16 $\frac{1}{4}$	16 $\frac{1}{4}$	4	4	15	15	4	4		1 $\frac{1}{4}$
4	22	14 $\frac{1}{4}$	14 $\frac{1}{4}$	3	3	15 $\frac{1}{4}$	15 $\frac{1}{4}$	3	3	1 $\frac{1}{4}$	
5	24	18	21	3	3	21	21	3	3	3	
6	17	18	18	3	3	18	18	3	3		
7	27	16 $\frac{1}{2}$	16 $\frac{1}{2}$	3	3	18	16 $\frac{1}{2}$	3	3	1 $\frac{1}{2}$	
8	19	16 $\frac{1}{2}$	16 $\frac{1}{2}$	3	3	15 $\frac{1}{4}$	15 $\frac{1}{4}$	3	3		1 $\frac{1}{4}$
9	20	23	23	3	3	21	21	3	3		2
10	18	18	18	2 $\frac{1}{2}$	2 $\frac{1}{2}$	16 $\frac{1}{2}$	16 $\frac{1}{2}$	3	3		1 $\frac{1}{2}$
11	18	19 $\frac{1}{2}$	18	3 $\frac{1}{2}$	3 $\frac{1}{2}$	19 $\frac{1}{2}$	19 $\frac{1}{2}$	3	3	1 $\frac{1}{2}$	
12	28	15 $\frac{1}{4}$	15 $\frac{1}{4}$	3	3	18	18	4	4	2 $\frac{3}{4}$	
13	22	23	23	3 $\frac{1}{2}$	3 $\frac{1}{2}$	19 $\frac{1}{2}$	19 $\frac{1}{2}$	3	3		3 $\frac{1}{2}$
14	16	21	21	4	4	19 $\frac{1}{2}$	19 $\frac{1}{2}$	4	4		1 $\frac{1}{2}$
15	20	19 $\frac{1}{2}$	19 $\frac{1}{2}$	3	3	18	18	3	3		1 $\frac{1}{2}$
16	17	18	18	3	3	18	18	3	3		
17	26	18	19 $\frac{1}{2}$	4	4	19 $\frac{1}{2}$	18	3	3	1 $\frac{1}{2}$	1 $\frac{1}{2}$
18	21	15 $\frac{1}{4}$	14 $\frac{1}{4}$	7	3	16 $\frac{1}{2}$	14 $\frac{1}{4}$	7	3	1 $\frac{1}{4}$	
19	24	16 $\frac{1}{2}$	15 $\frac{1}{4}$	3 $\frac{1}{2}$	3 $\frac{1}{2}$	16 $\frac{1}{2}$	16 $\frac{1}{2}$	3 $\frac{1}{2}$	3 $\frac{1}{2}$	1 $\frac{1}{4}$	
20	16	18	18	4 $\frac{1}{2}$	4 $\frac{1}{2}$	16 $\frac{1}{2}$	16 $\frac{1}{2}$	4	4		1 $\frac{1}{2}$
21	21	19 $\frac{1}{2}$	19 $\frac{1}{2}$	4 $\frac{1}{2}$	4 $\frac{1}{2}$	18	18	4	4		1 $\frac{1}{2}$
22	17	18	18	3 $\frac{1}{2}$	3 $\frac{1}{2}$	18	18	3	3		
23	26	18	19 $\frac{1}{2}$	4	4	19 $\frac{1}{2}$	18	3	3	1 $\frac{1}{2}$	1 $\frac{1}{2}$
24	18	15 $\frac{1}{4}$	14 $\frac{1}{4}$	3	3	16 $\frac{1}{2}$	14 $\frac{1}{4}$	3	3	1 $\frac{1}{4}$	
25	24	16 $\frac{1}{2}$	15 $\frac{1}{4}$	3 $\frac{1}{2}$	3 $\frac{1}{2}$	16 $\frac{1}{2}$	16 $\frac{1}{2}$	3 $\frac{1}{2}$	3 $\frac{1}{2}$	1 $\frac{1}{4}$	
26	16	18	18	4	4	16 $\frac{1}{2}$	16 $\frac{1}{2}$	4	4		1 $\frac{1}{2}$
27	17	14 $\frac{1}{4}$	16 $\frac{1}{2}$	3	3	15 $\frac{1}{4}$	15 $\frac{1}{4}$	3 $\frac{1}{2}$	3 $\frac{1}{2}$	1 $\frac{1}{4}$	1 $\frac{1}{4}$

BEFORE TONSILLECTOMY.						AFTER TONSILLECTOMY.					
CASE	AGE	TENSION.		PUPILS.		TENSION.		PUPILS.		CHANGE.	
		Rt	Lt	Rt	Lt	Rt	Lt	Rt	Lt	RISE	FALL
28	15	21	21	3	3	18	18	3½	3½		3
29	16	15¼	15¼	2½	2½	18	18	3	3	2¾	
30	18	16½	15¼	3	3	15¼	15¼	3	3		1¼
31	23	16½	16½	2½	2½	16½	16½	3	3		
32	21	18	18	3	3	19½	19½	3	3	1½	
33	15	11½	10	8	8	12¼	11¼	8	8	1¼	
34	14	13¼	12¼	8	8	13¼	13¼	8	8		1¼
35	23	18	19½	3	3	16½	18	3	3		1½
36	33	16½	16½	3	3	18	18	3	3	1½	
37	14	19½	19½	3½	3½	18	18	3½	3½		1½
38	14	21	21	3	3	18	18	3	3	3	
39	16	10	10	4½	4½	12¼	12¼	3½	3½	1¼	
40	32	19½	19½	3	3	21	21	3	3	1½	
41	23	12¼	12¼	3	3	15¼	15¼	3	3	3	
42	23	18	18	2½	2½	19½	19½	3	3	1½	
43	32	19½	19½	2	2	21	21	2½	2½	1½	
44	17	15¼	15¼	3	3	15¼	15¼	3	3		
45	19	19½	19½	3½	3½	18	18	3	3		1½
46	17	18	16½	2½	2½	18	16½	2½	2½		
47	20	18	18	2	2	21	21	2½	2½	3	
48	19	18	18	3	3	15¼	15¼	3	3		2¾
49	18	18	18	3	3	16½	16½	3	3		1½
50	17	15¼	15¼	2½	2½	15¼	15¼	2½	2½		
51	22	23	23	3	3	21	21	3	3	2	
52	20	16½	16½	3	3	15¼	15¼	3	3		1¼
53	20	18	18	3½	3½	16½	16½	3½	3½		1½
54	23	16½	16½	4	4	18	18	3½	3½	1½	
55	23	19½	19½	3	3	19½	19½	4	4		
56	25	16½	16½	3	3	16½	16½	3	3		
57	25	15¼	15¼	3	3	15¼	15¼	3	3		
58	17	18	16½	3	3	16½	15¼	3	3	1¼	
59	22	16½	16½	4	4	18	18	3½	3½	1½	
60	28	16½	18	4	4	18	18	4	4	1½	
61	27	19½	18	3½	3½	18	16½	3	3	1½	
62	19	16½	16½	3	3	18	18	3	3	1½	
63	20	18	18	4	4	16½	18	4	4		1½
64	20	21	23	3	3	18	21	2½	2½	3	3
65	18	15¼	15¼	2½	2½	13¼	14¼	3	3		2
66	18	16½	18	3	3	18	18	3	3	1½	
67	27	21	19½	3½	3½	18	18	3	3		3
68	20	18	18	3	3	18	18	3	3		
69	31	16½	16½	2½	2½	15¼	15¼	2	2		1¼
70	18	18	18	2	2	16½	16½	2	2		1½
71	16	16½	18	2	2	18	18	3	3	1½	
72	33	13¼	13¼	3	3	14¼	14¼	3	3	1	
73	24	16½	16½	2½	2½	15¼	14¼	3	3		2¼
74	26	16½	19½	2½	2½	18	18	3	3	1½	1½
75	29	19½	23	3	3	18	22	3	3		2
76	17	16½	16½	2½	2½	15¼	14¼	3	3		2¼

BEFORE TONSILLECTOMY.						AFTER TONSILLECTOMY.					
CASE	AGE	TENSION.		PUPILS.		TENSION.		PUPILS.		CHANGE.	
		Rt	Lt	Rt	Lt	Rt	Lt	Rt	Lt	RISE	FALL
77	18	19½	18	4	4	15¼	15¼	4½	4½		4¼
78	16	15¼	16½	5	5	15¼	15¼	4	4		1
79	13	18	18	4	4	18	16½	3½	3½		1½
80	20	14¼	21	4	9	13¼	19½	4	9		1½
81	20	19½	19½	5	5	15¼	15¼	4½	4½		4¼
82	25	18	13¼	5	5	13¼	14¼	4	4		4¾
83	16	15¼	15¼	3½	3½	13¼	14¼	3	3	2	2
84	28	19½	19½	4	4	18	18	4½	4½		1½
85	21	22¼	26	4	4	19½	23	3½	3½		3
86	22	21	21	3½	3½	18	19½	3	3		3
87	19	14¼	15¼	4	4	12¼	12¼	3½	3½		3
88	20	16½	16½	4	4	15¼	16½	4	4		1¼
89	24	18	15¼	3	3	15¼	15¼	4½	4½		2¾
90	14	18	18	4	4	19½	18	3½	3½	1¼	
91	14	19½	19½	3½	3½	19½	21	4	4	1½	
92	14	19½	18	3	3	21	21	3½	3½	3	
93	67	16½	16½	3	3	18	18	3	3	1½	
94	17	19½	19½	3	3	15¼	18	3	3		4¼
95	23	16½	18	3	3	18	19½	2½	2½	3	
96	20	11¼	12¼	2½	2½	13¼	13¼	3	3	2	
97	19	15¼	16½	3½	3½	16½	16½	3	3	1¼	
98	17	21	19½	3	3	19½	21	3	3	1½	1½
99	26	14¼	15¼	2½	2½	15¼	15¼	3	3	1¼	
100	22	18	16½	3½	3½	16½	16½	3½	3½		1½

Highest Tension 26mm  
 Lowest Tension 10mm  
 Average Tension 17¼mm  
 Greatest Fall 4¾mm  
 Greatest Rise 3mm

## ASTEROID HYALITIS.<sup>1</sup>

BY DR. T. B. HOLLOWAY, PHILADELPHIA.

AT odd times one observes in the vitreous certain alterations that are most striking and characteristic, such as the deposition of cholesterin crystals and those snow-white globular opacities that Benson has seen fit to describe under the title of asteroid hyalitis. I am using the same title solely to identify the type of case under discussion and not because I desire to participate in the discussion as to whether the term "hyalitis" is a justifiable one.

Prior to Benson, D'Oench, possibly referring to the same opacities, stated that they presented a picture like that of an astronomic chart, but others have preferred to liken them to snowballs. With others, since observing the first case, I have always referred to them as snowball opacities of the vitreous, not only because of the marked resemblance to actual tiny snowballs, but also because of their similarity to the flower of the *Viburnum opulus sterilis*, the common snowball shrub so frequently seen in our gardens. As to the term "hyalitis," it is probable that the majority of pathologists would take exception to it, despite the opinion of Straub.

The vitreous conditions in the patients observed by me conform quite well to the descriptions given by previous observers. The opacities are globular, many ellipsoid in shape, dull white, and not glittering as is cholesterin. A few may show a slight projecting spur, but all seem to have an appreciable diameter. In the first patient seen by me several white strands were noted. Pollak also observed strands, and he attributed them to the exaggerated elliptical shape of the opacities, that is, they became spindle form; this type,

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<sup>1</sup> Read at meeting of American Ophthalmological Society, 1917.

however, must be quite unusual. In certain cases there is but the slightest movement of the opacities on various excursions of the eyes, while in others the movements are quite extensive, but, irrespective of the extent, the opacities do not settle to the bottom of the vitreous chamber but return approximately to their original positions. They may be present in certain portions of the vitreous or the whole structure may be studded with them. In the presence of a lesion of the choroid these opacities, while present, are not necessarily in that portion adjacent to the lesion, as in Case 2, where there was a decided lesion in the lower and outer portion of the fundus, and only the central portion of the vitreous was involved; that is, the portions of the vitreous adjacent to the inner coats were free from these opacities. In the marked cases, when light is reflected into the eye by the ophthalmoscopic mirror, the white dots can be seen at a distance of 6 to 12 inches in front of the eye. When using a +7 or +16 lens in the aperture of the ophthalmoscope, one is apt to think at the first glance that he is dealing with two different types of opacities. Those within focus present the appearance above described while the other opacities appear milky and ill defined, not unlike the flocculent appearance presented by phosphates in the urine.

As to vision, this is apparently not affected in patients with a moderate involvement, and probably only slightly so where the whole vitreous is abundantly filled with these opacities; the reduction in vision, if it exists, is certainly less than one would expect.

While this type of opacity most closely resembles cholesterolin, I am convinced that a few minutes' study under proper conditions would promptly lead to the conclusion that they are not cholesterolin crystals. This conclusion is based entirely upon clinical observation, for up to the present time, as far as I have been able to determine, none of these eyes has been examined pathologically, nor have any of these patients been subjected to careful and complete physico-chemical tests. I do not mean to infer that cholesterolin may not be present in certain quantities, but certainly the characteristic appearance of the snowball opacity is not due to cholesterolin as we are accustomed to seeing it.

Whether these opacities are formed during some stage of the same process, that is concerned in the production of cholesterin crystals in the vitreous, or as the result of some analogous process, only future clinical and laboratory work can determine. Because these opacities most closely resemble cholesterin, and also because the patients showing these two types of opacities present certain other clinical resemblances, it may be well to briefly refer to synchysis scintillans.

Statistics are rather variable as to the frequency of synchysis scintillans; Webster (1) has recorded 5 among 45,000 cases, while Westpfahl (2), in the most recent tabulation, records 40 among 65,000 cases observed in the Würzburg Clinic. In addition to cholesterin, tyrosin, margaric acid, and phosphates have been found in these cases. Whether the crystals more frequently present a golden or silvery appearance need not be dwelt upon. It is essentially a condition of advanced years, said to be more common in women, and has been ascribed to diseases of the liver, arthritis, alcoholism, syphilis, and arteriosclerosis; it has also been found in cases of albuminuria and diabetes. Kipp (3) has described 6 cases, in 5 of which there was middle-ear disease. As to the eye, synchysis scintillans has been observed in association with signs of inflammation involving all parts of the uveal tract, affections of the retina, pathologic conditions of the lens, changes in the optic nerve, and glaucoma, and with certain corneal changes. Roemer has referred to its frequency in traumatic cases, 24 out of 50—48 per cent. In the 40 cases referred to by Westpfahl, 19, or 47 per cent., occurred in normal eyes or with unimportant affections of the cornea; the average age being sixty-two. In 10 cases, 25 per cent., there was associated cataract, the average age being seventy years. He believes this frequency accidental rather than causal. In 11 cases, 28 per cent., there were associated intraocular diseases, such as glaucoma, choroiditis, disease of the retina, optic atrophy, and traumatic detachment of the retina. The average age was sixty, although the youngest patient was twenty-five. Here again there appeared to be a causal relationship, but at least in the majority of cases it was not regarded as more than a coincidence.



The literature relating to these snow-like opacities is scanty, and while I have not exhaustively searched the literature of synchysis scintillans and thus probably have missed certain cases, I have found the following on record:

The first was one of four instances of synchysis scintillans referred to by Webster in 1884, and in certain respects I regard it as one of the most important. The patient was a woman seventy-six years of age. V. R. E. =  $\frac{3}{200}$ ; L. E. =  $\frac{20}{200}$ . Patches of xanthelasma were present about the internal canthus of each eye. The lenses showed incipient opacities. Studding the whole vitreous of the right eye were small, round, white opacities, and between these could be seen a few small glittering crystals.

In 1889 Valk (4), after alluding to an observation made by D'Oench, referred to two patients so affected:

CASE 1.—Male, aged seventy-three years. Complained of spots before his eyes and gradual failure of vision for five years. There was no history of injury. The vision of each eye was  $\frac{20}{40}$ , with and without correction.

CASE 2.—Male, adult. Detachment of the retina of the right eye, the result of trauma six months before coming under observation. The left eye had a convergent paralytic squint, the result of a blow fifteen years before. The vision was poor, and the eye was subject to recurrent inflammatory attacks. In the left eye of both of these patients the vitreous exhibited numerous white, glistening spots that appeared to be suspended and did not sink to the lower parts of this structure when the eye was stationary. Excursions of the globe produced slight movements of these white opacities, but some of them seemed to be held in place by minute dark filaments that were lost in the substance of the vitreous. The opacities appeared as round bodies involving all portions of the vitreous; they were intensely white and were surrounded by a dark circle, like an air-bubble under the microscope.

In 1894 Benson (5) referred to a male patient, aged sixty-two years, who had contracted syphilis thirty years before coming under observation. His general health was good, and examination of the heart and kidneys negative. There was no history of gout or rheumatism. The vision of each eye was  $\frac{6}{9}$ . No changes were noted in the right lens or fundus, but studding the whole vitreous were hundreds of small,

smooth, fixed, cream-colored spheres that in appearance resembled the stars on a clear night. During a period of nine months no change was noted in their appearance.

Argyll-Robertson stated he had observed an exactly similar case except that the bodies were movable and resembled a series of snowballs.

In 1896 Pollak (6) placed on record the data of a similar case observed in the Fuchs clinic. The patient was a female, aged fifty-one years. She had a high hyperopia and gave a history of an attack of acute articular rheumatism at the age of thirty. Both eyes were glaucomatous, the right being blind as the result of this process. In the periphery of the right eye there were areas of atrophy and choroiditic foci. A pigmented strand extended from the lower portion of the retina into the vitreous. In the vitreous of the right eye were numerous round and elliptic white opacities, and some of these were so much elongated that they might be referred to as cords or strands.

In 1909 Wiegmann (7), in discussing certain phases of synchysis scintillans, referred to a male, age fifty-seven years, with arteriosclerosis and profuse retinal hemorrhages of the right eye. The fundus of the left eye was said to be normal. By direct examination with gaslight, dull, snow-white particles could be noted in the vitreous of each eye that gave the impression of falling snow as the eye moved. He believed that calcium salts were probably the principal components of the crystals.

In January, 1917, Stark (8) reported the histories of three patients similarly affected:

CASE 1.—Male, aged sixty-four years. The optic nerves were rather pale, but there were no changes in the choroid or retina. Perimetric examination revealed a central scotoma for red and green. Albumin and granular casts were found in the urine and the Wassermann test was weakly positive. Vision could be improved to  $\frac{2}{30}$ . The disk and field changes were first attributed to the kidneys, later to syphilis.

CASE 2.—Alcoholic male, aged sixty years. V. R. E. = counting fingers at 12 feet; L. E. =  $\frac{2}{30}$ . The right nerve was pale. In this patient the opacities were noted only in the temporal portion of the vitreous of the right eye. The

poor vision of this eye was attributed to amblyopia exanopsia and phlyctenular macula of the cornea.

CASE 3.—Female, Mexican, aged thirty-five years. There were no intraocular changes except the characteristic vitreous change. The urine and Wassermann tests were negative. The corrected vision was  $\frac{2}{15}$  in each eye.

In addition to these cases several other references should be mentioned. Thus, D'Oench's (9) patient, referred to by Valk, had a detached retina. "The peculiar feature of this case, however, was found in the presence of numerous small white dots scattered throughout the detached portion, but most numerous near the periphery, apparently of the size of a pinhead or less in the ophthalmoscopic image, and not confluent." They resembled the stars in appearance. These dots Valk supposed to be in the vitreous, although D'Oench's statement is as above quoted.

Hill Griffith (10), in speaking of synchysis scintillans, states that in many of the cases he has seen opacities that appeared as white, glistening, round disks with the surface presented toward the observer. These shifted but little on movements of the globe.

The cases observed by me were as follows:

CASE 1.—Male, aged seventy-four years. This man was a patient of Dr. de Schweinitz's, and was seen by me August 19, 1909, during his absence from the city. The man complained of failing vision. V. R. E. =  $\frac{5}{60}$ ; L. E. =  $\frac{5}{50}$ . The right lens exhibited incipient opacities. The whole vitreous was studded with an enormous number of small, white globular opacities that moved freely with the movements of the eye, but did not settle to the bottom of the vitreous chamber. Several short white strands could also be seen, and on these were several white opacities, smaller than the average isolated opacity. The disk was healthy. In the periphery of the fundus there was a tendency to vague pigment blotches. The left eye did not show the characteristic vitreous changes, but otherwise was similar to the right eye. The corrected vision was  $\frac{5}{15}$  for the right eye and  $\frac{5}{12}$  for the left. As this patient lived in one of the western States and was under observation but thirty-six hours, no opportunity was afforded for general examination. He stated that he had had casts in his urine for the past twenty years.

CASE 2.—Male, aged fifty years. First seen September

19, 1912, when he complained of aching pain in his eyes, associated with flashes of light. The examination of the right eye was negative. In the left eye, located in the central part of the vitreous, were a number of these characteristic opacities. Far out in the lower temporal periphery was an isolated patch of retinochoroidal disturbance, the finer vessels near it showing considerable tortuosity and kinking. A few tiny hemorrhages and minute dilatations of the fine twigs could be noted. Between the inferior temporal artery and vein there was an elliptic area studded with tiny thin hemorrhages. V. with correction: R. E. =  $\frac{5}{4}$ ; L. E. =  $\frac{5}{4}$  -.

When about twenty he had alopecia areata. He had been troubled more or less with gastro-intestinal disturbances, and two years before had what he described as "ptomaine poisoning," and was quite ill for two and a half months; in fact, he did not fully regain his health for a year and a half. He has been compelled to avoid fruits and sugars, as they produce a diarrhea. He was requested to see his family physician, Dr. Charles Codman, who reported the lungs, abdomen, and heart to be negative. The urine was likewise negative, except for a few uric-acid crystals. Blood-pressure was 110-80. He was advised to take very small doses of sodium iodid. Later he was put on inunctions. He then passed from my observation until November, 1916, when he returned for refraction. At this time the opacities were certainly more numerous and more movable. No free hemorrhages were noted, but the same tiny aneurysmal or venous dilatation could be noted near the peripheral lesion. V. R. E. =  $\frac{5}{8}$ ; L. E. =  $\frac{5}{8}$  -. In February, 1913, he consulted Dr. Codman owing to redness of the tip of the nose. Over the affected area there was distinct capillary dilatation that persisted for some time. Blood-pressure was 135-110. In March, 1917, he again consulted Dr. Codman, complaining of feeling tired and weak, which he attributed to overwork. The urine showed a trace of albumin and also some indican.

CASE 3.—Female, aged fifty-five years. First seen by me on Dr. de Schweinitz's service at the University Hospital, April 7, 1913. Fundus examination of the right eye did not show any characteristic vitreous changes. The disk was slightly off color. In the macular region there was a yellowish choroidal lesion and three well-marked hemorrhagic areas of moderate size. Scattered about the posterior pole were a number of punctate yellowish lesions. There was a slight sclerosis of the vessels. Examination of the left eye revealed a number of these characteristic opacities in the vitreous. Small and old choroiditic foci were present be-

tween the disk and macula, and along the superior and inferior temporal vessels. V. R. E. =  $\frac{1}{150}$ ; L. E. =  $\frac{6}{8}$ , corrected. She was referred to the Medical Dispensary for examination, and the late Dr. Klaer reported that there were no signs of pulmonary tuberculosis and no evidences of specific disease. The symptoms were regarded as due to gastric or digestive disturbances and a very bad condition of her mouth and teeth. A later report stated that there was no nephritis; the blood-pressure was low and the Wassermann test negative. There was a faint trace of albumin in the urine.

CASE 4.—Female, aged seventy-three years. First seen at the University Hospital on March 23, 1917, owing to a slight conjunctival catarrh. Each eye showed a most extensive involvement of the whole vitreous with these characteristic opacities. Many of them showed irregularities or spurs and had a wide range of movement upon rotations of the globe. It was difficult to study the fundi, but certainly no disk changes were present and no lesions of the choroid or retina were noted. Each lens, however, showed incipient opacities. The correction of a high myopic astigmatism gave  $\frac{6}{7.5}$  vision for each eye. She was examined at the Medical Dispensary by Dr. Thomas Kelly, who reported that there were no demonstrable cardiac lesions and that the urine was negative, as was the functional kidney test. There was an arthritis of some of the smaller joints, and the patient complained of myalgic pains. There were no gastrointestinal symptoms aside from constipation. The Wassermann test was negative.

Of these 13 patients, 8 were males and 5 females; 11 were over fifty, 4 of these being over seventy; 1 thirty-five, and 1 was an adult, the age not being stated. In 2 instances there was bilateral involvement; in 7 the right eye only was affected, and in 4, the left eye. As to syphilis, 1 acknowledged the disease, 1 had a positive Wassermann, and in 3 this test was negative; all were presumably serum test. The associated ocular conditions have included lenticular opacities, signs of inflammation of the uveal tract, optic atrophy, and glaucoma, and, if D'Oench's patient be included, detachment of the retina.

From the above cases it can be seen that there is a striking clinical resemblance between these cases with snowball opacities and those showing cholesterol crystals. Both occur most frequently in individuals well advanced in years, and in

connection with essentially the same systemic conditions; both have been associated with varied and similar ocular disturbances or have been present without other visible intraocular or fundus conditions. Such being the case, it does not seem irrelevant to make brief further reference to synchysis scintillans. Sgrosso, in discussing this condition, refers to three groups: first, scintillation in the lens in the presence of cataract; second, exclusively in the vitreous, with integrity of the lens; third, involving all or some of the ocular cavities, with evident changes in the uveal tract. The cases of snowball opacities above mentioned would be grouped under the second and third division of this classification.

Concerning the pathogenesis of synchysis scintillans I would refer to Sgrosso's (11) work or to Roemer's contribution in the French Encyclopedia of Ophthalmology, vol. vi., which includes all the most important of Sgrosso's observations. Concerning group 1, above referred to, I would also refer to the contribution of J. Burdon-Cooper (12) on the hydrolysis theory in the pathology of cataract. He believes the tyrosin content of the aqueous acts as an indicator to the opacification of the lens, and also suggests that cholesterin may be but a further stage of the same process that leads to the production of tyrosin. In cataract associated with albuminuria and glycosuria the tyrosin in the lens and aqueous was much increased. Not less than a half-dozen different theories have been advocated to explain certain cases falling in group 2 above referred to. As to group 3, it seems that in these cases some other factor must be required aside from the lesions with which this phenomenon has been associated, otherwise why the infrequency of the condition as compared to the enormous number of cases showing similar fundus changes?

What is the relation of hypercholesterolæmia to the type case immediately under discussion? Diseases of the gall-bladder, arteriosclerosis, nephritis, diabetes, pregnancy, and some phases of certain infectious diseases may all give rise to an increase in the cholesterol content of the blood. Reh-fuss, who has devoted much attention to these cases of cholesterolæmia, advises me that he has not seen instances of

vitreous changes such as I have referred to, although he has not carefully examined the vitreous in each instance while using the ophthalmoscope in his routine clinical work. Even so, it is difficult to see how they could be overlooked, even in a most casual examination, if they were present.

In speaking of diabetic lipæmia, von Fürth (13) quotes Klemperer as stating that the condition is really a lipoidæmia, the bulk of the ethereal extracts consisting of cholesterin and lecithin and not of fats. He quotes Magnus-Levy as stating that the fats fail to pass out of the blood-stream either because of some change in the permeability of the walls of the capillaries or from lack of favorable condition in the fats. What these are it is not possible to say. I have not reviewed the literature of retinal lipæmia that usually develops in young subjects and is apt to be a late manifestation, but in the few cases I am aware of no such characteristic changes were observed. As a matter of interest, mention might be made of the presence of xanthelasma in Webster's patient, a condition which has been attributed to cholæmia and regarded by Chauffard and Laroche (14) as resulting from a transitory or permanent hypercholesterolæmia.

Concerning these snow-like opacities, I believe they constitute a manifestation which, like cholesterin, is most frequently seen in advanced years, and that it is not congenital, as has been suggested. That the condition is or may be progressive, is shown by my second patient, who has been under observation about five years. At first the movement is slight, but doubtless becomes more marked as the condition progresses. In Cases 1 and 6 the opacities had the widest range and were abundant. Cases 2 and 3 had few opacities and less movement. While it may be a coincidence, the opacities in these last two cases seemed to be more perfectly formed.

Considering the clinical resemblance of these cases to those showing cholesterin crystals of the character we are accustomed to see, does not Webster's case, where these two types of opacities were seen side by side, still further suggest the belief that the snow-white opacities are formed during the same or similar process that has to do with the deposition of cholesterin in the vitreous?

All observers who have written concerning these opacities have agreed that they are not cholesterin, although I would not be surprised if cholesterin would be found in an analysis of the vitreous of one of these patients. Whether calcium salts enters into their composition, as suggested by Wiegmann, can be proved only by opportunities that so far have not been available, and as to the pathogenesis, this must remain even more obscure than is that of the formation of cholesterin in the vitreous. Concerning Wiegmann's suggestion, it is interesting to note that in a case of synchysis scintillans Königstein (15) found crystals that resembled the oxalate and sulphate of lime obtained by Panas in experimental naphthalin cataract.

Finally, while these cases are not frequently encountered, I believe they are more common than the literature indicates. It is probable that in some instances they have been grouped with the true cases of synchysis scintillans. They should not be confused with the circular or disk-like opacities sometimes seen in severe cases of uveitis and that resemble in appearances the large mutton-fat drops seen on Descemet's membrane. I hope to be able to report further concerning Case 6 and probably Case 3.

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ILLUSTRATING DR. COHEN'S ARTICLE ON LEUKÆMIC MANIFESTATIONS IN THE EYE: A CLINICAL AND PATHOLOGICAL REPORT

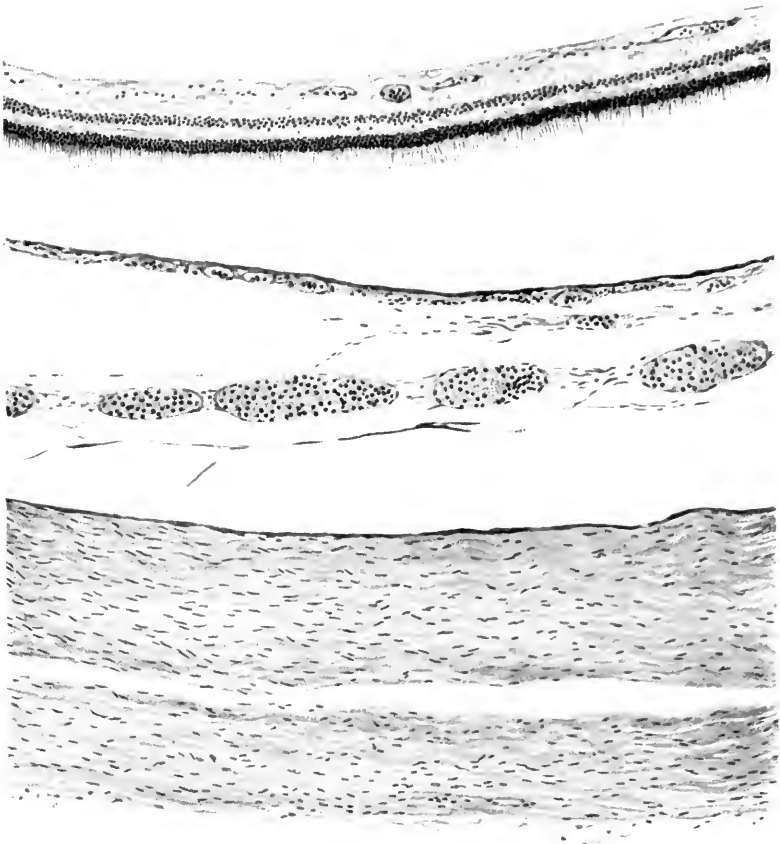


FIG. 1.

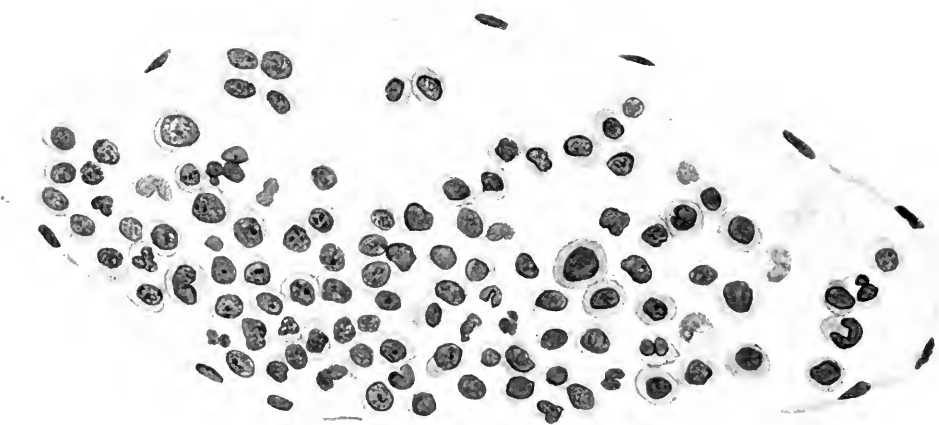


FIG. 2.

## LEUKÆMIC MANIFESTATIONS IN THE EYE: A CLINICAL AND PATHOLOGICAL REPORT.<sup>1</sup>

BY DR. MARTIN COHEN, NEW YORK.

*(With two figures on Text-Plate V.)*

Mr. B. H., a laborer 23 years of age, was admitted to the Harlem Hospital suffering from lobar pneumonia. The usual pulmonary signs were present and, in addition, the patient presented clinical findings which were indicative of his blood condition. The liver extended four fingers' breadth below the costal margin, and the spleen to the crest of the ilium. There was no enlargement of the superficial lymph nodes. The blood showed a marked anæmia: the hæmoglobin was 45 per cent., and the red cell count 1,800,000. The leucocytes numbered on two occasions 485,000 and 295,000, and showed the following proportion: Number of cells counted, 400; polynuclears, 43 per cent.; transitionals, 3 per cent.; lymphocytes, 12 per cent.; large mononuclears, 5 per cent.; eosinophiles, 2 per cent.; mast cells, 2 per cent.; myelocytes, 33 per cent. (neurophilic myelocytes, 22, eosinophiles, 7, basophiles, 4). In addition, there were a large number of nucleated red blood cells.

The pneumonia did not resolve and the patient died after twelve days.

The post-mortem report by Dr. Gonzales confirmed the clinical findings. The liver weighed 4.1 kilos, and the spleen 2.3 kilos,—both organs being more than twice the normal weight. Microscopical examination of these organs showed the typical engorgement of blood and lymph spaces with myelocytes, and in places also masses of megakaryocytes; the bone marrow was chocolate color and its markings were indistinguishable. On microscopical examination, masses of myelocytes and megakaryocytes were seen. The

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<sup>1</sup> Presented at the January meeting of the Section on Ophthalmology, New York Academy of Medicine. 1917.

clinical and pathological diagnosis was: Chronic Spleno-Myelogenous Leukæmia and Lobar Pneumonia.

The eye examination was last made five days before death, and presented the following appearances:

The palpebral conjunctivæ were extremely pale; otherwise external examination was normal. The acuity of vision, field of vision, intraocular tension, and media were normal. The irides were markedly pale and showed an ectropion of the uveal pigment at the sphincter margin of iris. The pupillary light reaction was sluggish.

The ophthalmoscopic examination was practically the same in both eyes. The fundus color was distinctly lighter than one sees in a normal eye-ground. The disk was of a dirty white color; its margin was completely blurred and was moderately elevated above the surrounding retina. The retinal veins were characteristically tortuous, moderately and uniformly dilated, and of a darkish red color; the retinal arteries were slightly tortuous, of normal caliber, and showed clearly the central light-reflex streak. A circular isolated retinal hemorrhage was seen in the right eye; its size was one-third the diameter of the disk and it was situated two disk diameters to its nasal side. No other pathological changes were present in fundus. *Diagnosis* of fundus condition: Papillo-Retinitis. *Etiology*: Spleno-Myelogenous Leukæmia.

*Microscopical Report* of the eye: One eye was enucleated ten hours after death, fixed in Müller's fluid, then embedded in paraffine, and finally sectioned. The sections were then stained in a Giemsa solution in order to better study the individual intravascular cellular element of the tissues involved.

The cornea and media are normal. Iris structure is markedly atrophied, showing little stroma tissue; the pigment epithelium is hypertrophied and an ectropion of the pigment is apparent. Schlemm's canal is empty. Lens is normal, but there is seen a deposit of uveal pigment on the anterior capsule at its central area. Ciliary processes are atrophied; ciliary body is normal. The choroid shows characteristic changes, especially posteriorly where the veins are decidedly dilated (see Fig. 1) and filled with numerous leucocytes (neutrophilic myelocytes, eosinophiles, polynuclears), and a few erythrocytes (see Fig. 2). A large hemorrhage is seen in Haller's layer. The marked dilatation of the choroidal veins is probably an early manifestation of choroiditis; a similar venous dilatation exists in other organs of the body. In the retina, the identical intravascular changes are present, but to a less degree, owing to its vascularity, and there is seen a hemorrhage situated in the

nerve-fiber layer of the retina; the macular region is slightly oedematous.

The papilla is moderately swollen, indicating a papillitis. The optic nerve and sheaths are normal; its veins contain similar cellular elements to those found in other structures of the eye.

*Microscopical Diagnosis:* Neuro-Chorio-Retinitis.

REMARKS.

1. It is apparent from this report that the changes occurring in the eye in spleno-myelogenous leukæmia are analogous to the changes manifest elsewhere in this disease.

2. The pathological condition is concerned mainly with the intravascular elements which characterize this blood state, as has been noted by other authors.

3. The involvement of the choroid and retina points to the existence of a Chorio-Retinitis Leukæmica instead of a Simple Retinitis Leukæmica as generally described.

## PENETRATING INJURY LIMITED TO THE EYEBALL FOLLOWED BY ACUTE TETANUS.<sup>1</sup>

BY DR. ROBERT SATTLER, CINCINNATI, OHIO.

THE following excerpts have been taken from a complete clinical history of a case of acute tetanus, following a penetrating wound of the eyeball, furnished me by Dr. Shanks, interne of the Ophthalmic Department of the Cincinnati General Hospital.

C. H., æt. 21, farmhand; while seated on the rear end of a wagon on which he was riding, his right eye was struck and perforated by a punch shaped arrow of desiccated horsetweed which had been aimlessly shot from a crossbow by a boy on the opposite side of the street, a distance of fifteen or more feet.

The implanted foreign body was speedily jerked out of his eye by his own hand and he sought at once the advice of a nearby physician, who removed several smaller pieces of foreign substance, but whether these were fragments of weed or dirt he was unable to state.

At the Cincinnati General Hospital he was examined immediately after admission, or thirteen hours after the injury was received, and it was found that the entrance perforation or wound was strictly limited to the cornea with mangled iris tissue and lens substance protruding. Under local anæsthesia the wound was cleared of all extraneous matter and shreds of iris tissue and lens substance removed. Great prostration and pain, œdema of the eyelids and bulbar conjunctiva, and an opalescent discharge pointed to an imminent vicious reaction. The further statement that the improvised weed arrow had been just pulled out of the ground by the boy who shaped and shot it, induced me to advise and direct immediate enucleation. This was done on the same day by Dr. Ray, assisted by Dr. Shanks.

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<sup>1</sup> Read at Meeting of Am. Ophth. Soc., Hot Springs, Va., 1917.

Nothing noteworthy happened until the afternoon of the third day, following the operation, but in the meantime the patient had been up and about, and was apparently in excellent health and spirits and had not complained of pain or discomfort and the cavity appeared clean and free of all irritation and undue secretion. He referred to difficult mastication and stiffness of the muscles of the jaw, more marked on the right side. From further examination and consultation it was evident that trismus was already present. He was given a large dose of antitetanic serum intravenously and removed to the serological department in charge of Dr. O. Berghausen, who from this time on commenced the liberal and systematic administration of antitetanic serum. In spite of large and sustained doses injected intraspi-  
nally, 10,000 units once a day, and three hourly ones of 3000 units each, intravenously,—in addition, the orbit was exten-  
terated, under general anæsthesia, and this followed by constant local applications of the serum, and antitetanic serum was injected into and along the sheath of the optic nerve,—the patient died on the seventh day after admission.

The autopsy disclosed: congestion of the lungs, acute dilation of the heart, fatty degeneration of the liver with passive congestion of left lobe, acute splenic tumor, cloudiness of the meninges, and oedema of the brain.

From this brief recital of the principal points it can readily be inferred that we were led to precipitate radical surgery upon the very heels of the injury to avert the dangerous consequences of a next to certain panophthalmitis. We did not lose sight, however, of the fact that the penetrating character of the injury and the more than probable intraocular lodgment of extraneous or even contaminated soil matter included also tetanus among the possible remote complications.

We shared the hope that prompt enucleation of the globe and the very short time interval (24 hours) which elapsed between the happening of the injury and removal of the eye would insure safety against immediate and more remote and only possible dangers. From a cursory review of the literature of modern conclusions of acute tetanus, following injuries of the eye and orbit, it was found that these have been promiscuously grouped under cephalic tetanus. It was impossible for me to determine from the sources accessible to me whether this included a single case in which the injury was strictly limited to the eyeball. The search made, established only that the

larger number were combined ones of the eye and tissues of the orbit or in which missiles or penetrating forces had inflicted other similar wounds to adjacent regions of the face and head. Nor has my own experience furnished me with a similar example of an injury limited to the eyeball, which was followed by acute or chronic tetanus.

The period of incubation of tetanus is necessarily vague in every case. It was unusually short in the present one. It might lead us to assume that owing to the rapid diffusibility of the toxins of the tetanus bacilli, it may have been incredibly short or next to coincident with the injury itself, in this or similar cases. This observation, if confirmed, may in the future direct in every case of this class the early and more liberal use of prophylactic doses of antitetanic serum or long before definite symptoms of trismus are unmistakably present.

Reviewing briefly the probable, or at present accepted bacteriologic facts (1), and the limited clinical ones with case reports of ocular and orbital injuries followed by tetanus which support the former, it would appear that if a destructive pyogenic reaction results, this may prove a possible safeguard against an otherwise certain fatality if, in addition, prophylactic and therapeutic doses of acute antitetanic serum are also promptly and liberally administered.

Brons (2), in a review of infectious lesions of the eyelid and orbit, refers to three cases of tetanus following injuries of the eyelids and orbit. Two were gunshot injuries. In the first reported by Jess (3), the eye was completely dislocated at the time of the accident and was enucleated. Orbital abscess followed with presence of tetanus bacilli, confirmed by culture and their toxins pathogenic to animals. Tetanus did not develop, but vigorous and large doses of antitetanic serum were resorted to.

In Flacher's (4) case trismus or head tetanus followed an orbital abscess, specific organisms were present, cultures positive. Energetic serum treatment in this case also resulted in recovery. A third case reported by Salus (5), penetrating wound and abscess of orbit with implantation of the terminal end of a wooden switch, ended in death, in spite of liberal and systematic serum treatment.

Zurnedden (6) in an exhaustive review of the bacteriology,



etc., of infectious diseases of the eye does not mention tetanus resulting from injury limited to the eyeball.

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A CASE OF UNILATERAL PROPTOSIS. EXPLORATORY OPERATION. TENTATIVE DIAGNOSIS OF AN ETHMOIDAL OSTEOMA.<sup>1</sup>

BY DR. ROBERT SATTLER, CINCINNATI, OHIO.

THE following clinical history refers to a recent case of supposed neoplasm of the orbit in which surgical interference became imperative, for the reason that, to a moderate degree of transitory left-sided proptosis, which had been present for two years but without discomfort or impairment of vision, was suddenly added greater prominence with persistent irritability of the eye, passive congestion of lids and superficial structures, and considerable œdema. Upon his second visit to me, two weeks after the first, vision, which at that time was full normal, had during this short interval dropped to  $\frac{20}{30}$ , but without adequate changes in the eye ground, other than moderate venous stasis and slight discoloration of the optic papilla.

The disclosures of a most searching exploratory operation conclusively upheld the assertion that a tentative diagnosis of the early stage of an osteoma having its origin in one of the posterior ethmoidal cells or even of the sphenoid, was its only justifiable inference.

This was the more disappointing as the nature and clinical characteristics, their concealed location and inexorably slow growth, and in particular the greater mortality attending surgical interference, of these bony growths, so often of ivory hardness, are all well-known facts of common experience. For these reasons it was decided to await further developments and to add in every way to the comfort of the patient and to prepare the affected region for the best possible subsequent

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<sup>1</sup> Read at meeting Am. Ophth. Soc., Hot Springs, Va., 1917.

inspection and observation, and for the present to defer radical surgery. In order to permit readier access to the apex region of the orbit, it was necessary at the time of the exploratory operation to remove the globe as this alone enabled us to examine and confirm with accuracy the protrusion of the still intact inner and upper wall of the cavity. The larger wound of the exploratory operation healed promptly and the traumatism resulting from the more or less rude handling of the tissue owing to the longer time required for the exploratory operation also disappeared rapidly. The patient now wears an artificial eye and there has been no observable change in the deeper regions of the affected orbit.

A former experience with a similar case, the exploratory operation preceded by enucleation of a blind globe, disclosed the presence of a rounded knob of hard consistency about the size of a hazel-nut in the extreme depth of the orbit. It was assumed that the osteoma had started in one of the posterior ethmoidal cells and had forced its way into the orbit. It was subsequently discovered that this rounded mass which was removed at the first operation was the terminal projection of an osteoma which took its attachment from the anterior wall of the frontal sinus and had pushed its ruthless compression directly backwards towards the apex of the orbit. At the age of 27 it had caused absorption of the orbital wall and was far outside its original starting place. At a later date, the frontal sinus was fully exposed and the inner orbital wall ablated and the entire tumor exposed and removed. In the present case a series of carefully prepared X-ray plates since the exploratory operation have been taken by Dr. Lange, but have afforded us no proof of the presence of a more marked shadow of the deeper recesses, supposedly the seat of the tumor.

For these reasons a positive diagnosis is held in abeyance and major surgical interference will be deferred until the needs call for this, either that pain becomes unbearable or other complications arise, or until the tumor through its growing dimensions becomes more accessible to inspection and palpation and more readily amenable to surgical treatment.

The patient was a lad of 14, whose splendid physique and exuberant spirits suggested excellent health in the past

and present and this was fully confirmed by his own and his parents' statements with the exception that for long periods he suffered severe and persistent headaches.

For about two years, members of his family have noticed a slight prominence of his left eye but with no complaint on his part either of impairment of vision, or other discomfort. In the beginning the prominence was transitory. His athletic pursuits were at first held responsible for this, but careful observation disproved it. Suddenly two months prior to his first visit to me, and without assignable cause, overexertion or traumatism, the protrusion of the left eye became more pronounced and permanent.

Present condition: Marked left-sided proptosis, directly forwards and outwards or parallel to the axis of the orbit; unrestricted muscular excursions; objective and ophthalmoscopic examination discloses nothing noteworthy.  $V = \frac{20}{20}$ ; no limitation of visual field for white or colors. Examination of adjacent sinus cavities, frontal, ethmoid, sphenoid, aided by X-ray, transillumination, anterior and posterior rhinoscopy, failed in the discovery of an adequate cause for the displacement of the left eye.

Blood tests, as well as tests for lues and tuberculosis, also furnished no clue.

An exploratory operation was at once advised to determine the location and character of the tumor mass. It was furthermore stated as probable that if the neoplasm was within the funnel of the recti muscles that successful resection of the tumor mass might be accomplished with conservation of the globe.

These suppositions were all disproved by the exploratory operation two weeks later, the patient having suddenly and persistently experienced pain and discomfort with marked irritability of the eye owing to the less perfect protection of the cornea, which already showed slight haziness and irregularities of surface from superficial contusions. Vision was reduced to  $\frac{20}{80}$ , eye ground normal.

A roomy Kroenlein incision afforded access to and thoroughly exposed the septum orbitale. The fascia was opened close to and parallel to the lower and outer orbital margin. Some fat was removed to permit readier introduction of the finger. The adjacent floor, which region on transillumination showed a somewhat darker shadow, was first explored, followed by a similar thorough palpation of the outer wall. No trace of periorbital thickening or the presence of pathologic alterations could be discovered. The funnel of the muscles was next examined only to find the optic nerve intact and easily traced. This was followed by a deliberate examination of the roof and inner wall with

negative results. The eyelids, which had been provisionally closed, were now released and the globe easily dislocated forwards in order to permit an examination of the extreme apex region of the orbit. After careful search a rounded, extremely hard protrusion of the inner and upper wall was discovered with its convexity outwards and with perceptible contraction of the lumen of the orbit. This was confirmed by Dr. Ray and Dr. R. R. Sattler. We then decided to enucleate the globe in order to permit an easier access to the region, corresponding as best we could judge to the ethmoid or sphenoid bones, and from which the flattened hard button-like projection had pushed the orbital wall out and contracted its lumen. The improved means of access through the combined openings of the exploratory incision and the large opening of the conjunctival sac enabled us to confirm the presence of this rounded bulge of the inner wall and to compare its extreme hardness with the adjacent region forward.

After careful consideration of the gravity of the situation it was determined not to proceed with the removal of the small and probable osteoma, but to seek further information from carefully prepared X-ray examinations and the disclosures of further observations.

The septum orbitale and external wound were securely closed and the conjunctival sac sutured. Recovery was speedy and uneventful.

## SARCOMA OF THE CHOROID WITH SECONDARY CHANGES.<sup>1</sup>

BY DR. EDWARD JACKSON, AND DR. WILLIAM C. FINNOFF,  
DENVER, COLO.

THE following case is of especial interest on account of its long duration, the time the eye was retained after it reached the stage of secondary glaucoma, the violent orbital inflammation that led to its removal, and the significant changes revealed by the anatomic study of the globe.

L. L., a ranchman, gave the following history. He had observed the field of vision of the left eye gradually lost, beginning with the upper nasal portion. August 9, 1902, he consulted Dr. Harold Gifford, of Omaha, to whom (and to his associate, Dr. J. M. Patton) I am under obligations for notes of the case. "The vision of the right eye =  $\frac{2}{3}$ ; left = light perception. There was extensive retinal detachment of the left eye above and around the whole periphery below. He gave a history of having been struck in the forehead by a horse's head some time previous. There was no notation of his having any muscular disturbance at that time."

Mr. L. also gave the history of a fall on the back of the head; and suggested that his trouble might have come from "sleeping out and getting chilled." Soon after seeing Dr. Gifford the eye began to turn in, and the deviation gradually increased until it reached the present condition. On March 22, 1910, he was seen by Dr. George L. Strader, of Cheyenne, who kindly informs me that he noted detachment of the retina and complete blindness of the left eye, and gave glasses for the right. After this no apparent change occurred in the eye until about one year ago, when it began to pain him occasionally and to get somewhat red.

January 26, 1915: *Present Condition:* A healthy man,

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<sup>1</sup> Read at meeting of Am. Ophth. Soc., 1917.

aged fifty. Right eye, appearance normal, media clear, vessels in good condition, fundus normal. V. with sph. +1=1.2.

Pupils circular; right 3mm, left 4mm in diameter. Both react well to light thrown in the right eye; not at all to light thrown in the left.

Left eye has no light perception. It converges 50 centrad, but moves freely in all directions. The anterior perforating veins are dilated. There is a thin, band-like, subepithelial opacity of the cornea, extending horizontally to 1mm from each limbus and 5mm wide. There is gray opacity of the lens nucleus with clear cortex. Tension under holocain (Gradle-Schiötz tonometer): Right 9 (28mm), left 4 (52mm). Transillumination gives no pupillary glow through the temporal quadrant of the sclera, but it is good in all other directions.

The ophthalmoscope shows gray opacity of the lens nucleus with clear cortex. The pupil, dilated to 6mm with euphthalmin and cocain, shows red reflex in the periphery, upward, inward, and downward, but not outward.

January 27th: Under pilocarpin the eye has become entirely comfortable. Enucleation was advised, but not allowed.

February 6th: His eye remained comfortable until, on the third and fourth instant, he was exposed to cold on the range. On the fourth it became painful. The tonometer shows 4 (52mm). The eye is still hyperæmic and sensitive to light thrown in the right eye. It presents no point of ciliary tenderness, but there is slight tenderness when the globe is pushed back into the orbit. Pilocarpin contracts the pupil from 4.5mm to 2.5mm. The patient still declined enucleation, and was not seen again for seventeen months.

July 6, 1916: The eye remained comfortable under the use of pilocarpin until one week ago. Then it got sore and swelled. This followed getting overheated on the trail, and bathing his feet and legs in very cold water. The eye became painful that night. The lids are now greatly swollen, the eye pushed forward, the conjunctiva very oedematous and chemotic, with ecchymoses. The movements of the eye are greatly limited by orbital swelling.

The cornea presents a grayish-yellow ring 1mm within the limbus and 1mm wide. Within this ring the cornea is dark in color and quite opaque. There is rather free conjunctival discharge, smears from which prove negative.

July 7th: The eye was enucleated under ether. It was found tightly wedged into swollen orbital tissue to which the globe was tightly adherent and much of which was removed with the eyeball, especially the tissue around the optic nerve.

The enucleation was followed by free bleeding. There was little subsequent pain, and recovery was uneventful. On July 12th he left the hospital, ecchymoses and discharge diminishing. On July 14th all dressings were omitted.

He continues in good health and the orbit remains healthy. An artificial eye is worn with comfort.

*Macroscopic Examination.*—The globe was very hard. The cornea has a yellowish ring near the periphery, and the remainder of the cornea is opaque.

Anteroposterior diameter,  $24\text{mm}$ ; the transverse,  $24.5\text{mm}$ . The stump of the optic nerve is  $8\text{mm}$  long. The vitreous chamber is filled with a reddish-brown mass which resembled an old hemorrhage. The anterior chamber is filled with a whitish exudate. The anterior chamber is  $2\text{mm}$  deep. A  $2\text{mm}$  pupil. Anteroposterior diameter of the lens is  $5\frac{1}{2}\text{mm}$ ; transverse,  $9\text{mm}$ .

The globe was fixed in Zenker's fluid and embedded in celloidin. Sections were stained with hematoxylin and eosin, Mallory's connective-tissue stain, Van Gieson's stain, with iodine, and for iron.

*Microscopic Examination.*—The corneal epithelium varies in thickness. At the limbus it is five or six cells deep; this rapidly thins to a layer made up of one or two cells. In the center and in other areas there is a complete absence of corneal epithelium, and Bowman's membrane constitutes the anterior layer.

Bowman's membrane is finely granular, and is intact throughout. In it are several round and oval areas of degeneration which stain blue where hematoxylin and eosin are used; blue with Mallory's connective-tissue stain; red with Van Gieson's, and brown with iodine. They are more numerous where the epithelial covering of the cornea has been lost.

The substantia propria is infiltrated from the limbus to the center of the cornea with numerous flattened cells which show the various stages of degeneration. The infiltrate occupies the spaces between the corneal lamellæ. Near the limbus the cells infiltrate only the spaces in the posterior half of the substantia; at a point three-fourths of a millimeter from the limbus the infiltrate occupies the whole depth of the substantia. At this place they are more closely packed together in the spaces, occupying the anterior one-half of the corneal substance. The infiltrate occupies the whole depth of the substantia for about  $1\text{mm}$ , and then gradually confines itself to the anterior one-half of the cornea. This gives the section of the corneal infiltrate a triangular appearance, the base being the denser portion and the apex the thinner portion in the center. A ring  $1\text{mm}$  wide,



located about three-fourths of a millimeter from the limbus, corresponds to the very dense infiltration of cells which occupies the whole depth of the substantia. The ring resembles, histologically, the peripheral annular infiltrate which occasionally follows perforating wounds. The cells infiltrating the substantia have wandered so far from their source of nourishment that the majority have undergone degeneration and their identity is entirely lost. The nuclei are completely disintegrated. Only after prolonged search over several sections with the oil-immersion lens a few cells were found which had not been completely degenerated, and can be identified as polymorphonuclear leucocytes and lymphocytes. Blood-vessels have not proliferated into the cornea.

Near the center of the cornea the corneal lamellæ become swollen by œdema. Small areas of degeneration, which are similar in appearance and staining reaction to those found in Bowman's membrane, are seen scattered through the corneal lamellæ. Just under the epithelium, at the limbus corneæ, opposite the termination of Bowman's membrane, is a mass of hyaline degeneration.

Descemet's membrane is intact, and only an occasional corneal endothelial cell can be found on its posterior surface.

The epithelium of the limbus, cornea, and conjunctiva is invaded with numerous polymorphonuclear leucocytes. In the loose stroma of the limbus and the conjunctiva non-pigmented, spindle-shaped sarcoma cells surround the blood-vessels. This tissue is also infiltrated with polymorphonuclear leucocytes and lymphocytes.

In several sections Schlemm's canal is packed with tumor-cells. The meshwork of the iris angle is also filled with these cells, red blood-corpuscles, and polymorphonuclear leucocytes. The spaces which are not filled with cells are compressed by the iris, which has been pushed forward.

There is a pronounced peripheral anterior synechia of the iris to the cornea. The whole filtration angle is blocked, and the iris is adherent to the cornea for about 1mm.

The anterior chamber is filled with an exudate of fibrin, a few scattered polymorphonuclear leucocytes, and erythrocytes. Small round collections of pus-cells, resembling minute abscesses, are seen in the exudate. Near the iris the exudate has pigment-granules scattered through it.

The pupil is 2mm in diameter and is not occluded by any inflammatory membrane. The pupillary margin of the iris is covered with a mass of degenerated pigment epithelium and fibrin, which bows forward and results in a very striking ectropium uveæ.

Several of the iris crypts are filled with degenerated leuco-

cytes. The iris vessels are filled with blood, and several of them are surrounded by an infiltrate of leucocytes. The iris stroma is swollen by oedema. There are many pus-cells between the sphincter fibers. The pigment epithelium of the iris has undergone a very marked degeneration. Pigment-granules have wandered from the cells and invaded the posterior and anterior chambers and iris stroma. The cells with their nuclei have lost all their normal characteristics. Resting on the temporal one-third of the posterior surface of the iris is a pigmented mass which contains degenerated pigment epithelial cells, polymorphonuclear leucocytes, fibrin, and numerous pigment-granules. The protoplasm of the leucocytes in this area is filled with the pigment-granules. There is no synechia of the iris to the lens-capsule.

The epithelium of the ciliary body has undergone a more complete degeneration than that of the iris. The ciliary processes are devoid of epithelium. The blood-vessels are engorged with red cells and leucocytes. The stroma is hazy from oedema. Tumor-cells and leucocytes have invaded this structure and separate the fibers in many places. A cyclitic membrane extends from the ciliary body to the posterior surface of the lens. The exudate has undergone organization in many places. Capillary loops, fibroblasts, and connective-tissue are present. The exudate is invaded with leucocytes and pigment-granules.

There is almost complete degeneration of the retina; only near the optic nerve can a few nerve-fibers be found.

The choroid of the temporal one-half of the globe is one mass of sarcoma-cells. The tumor mass replaced the vitreous in two-thirds of the globe. Near the sclera the tumor-cells are well developed; but, as the middle of the globe is approached, the tumor changes in appearance and becomes a mass of degenerated cells. The cells in the degenerated portion of the tumor have lost their outline and many of their pigment-granules. The blood-vessels of this portion have shared in the degeneration, and hemorrhages have taken place, completely replacing the vitreous in the greater portion of the globe with red cells and fibrin. Occasionally a blood-vessel is found in the degenerated portion of the tumor, which is surrounded by several layers of new-formed healthy sarcoma-cells. In the tumor are many scattered polymorphonuclear leucocytes and lymphocytes. In some areas in the tumor and exudate dense collections of these cells are found. Around the blood-vessels of the sarcomatous choroid, near the sclera, are collections of lymphocytes. In some areas this infiltration is very dense. On the nasal side of the globe the choroid is not so thick, and a few areas resembling normal choroid can be seen. In the choroid

near the nerve there is an area of ossification. In the remainder, between the blood-vessels, are well-developed sarcoma-cells. The lamina vitrea on this side of the globe is intact and has many warts upon it. The pigment epithelium of the choroid has undergone a degeneration which is similar to that of the ciliary body and iris.

The sclera in many places is invaded with degenerated leucocytes similar to those seen in the cornea. The paravascular spaces of the anterior and posterior ciliary vessels and the vena vorticiosa are invaded with sarcoma-cells. The cells can be traced from the intraorbital tumor through these spaces to the orbital tissue.

There is a deep glaucomatous cupping of the nerve-head; the cup is three-fourths of a millimeter deep, and is filled with an exudate of fibrin and red blood-corpuscles. The vessel-walls in the optic nerve are thickened and the vessels are surrounded by a dense infiltrate of lymphocytes.

The vitreous is detached and pushed forward behind the lens by the tumor and hemorrhage. The lens is cataractous.

The tissue which adhered to the globe during enucleation is invaded with tumor-cells which surround the blood-vessels.

*Diagnosis.*—Spindle-cell melanosarcoma of the choroid, with metastasis through the globe into the conjunctival and orbital tissue. A mild panophthalmitis with a peripheral annular infiltrate of the cornea without perforation of the globe. A cataractous lens and a secondary glaucoma following a choroidal sarcoma.

The first striking feature in this case is its duration—over fourteen years from the time sight was noticeably impaired, and two and one-half years after glaucomatous symptoms had arisen until the eye was enucleated. It is certain that detachment of the retina had occurred when the eye was first seen by Dr. Gifford. That the detachment was due to the development of the sarcoma is probable. The impairment of vision began in a portion of the visual field corresponding to the situation of the growth and was gradually progressive. The patient's description of how the field was affected was spontaneous, definite, and obtained before the location of the growth had been determined. The history of other possible causes of detachment was such as might be obtained in any case of loss of sight.

That the diagnosis of sarcoma was not made earlier by competent men, like Dr. Gifford and Dr. Strader, does not militate against this view. The evidence upon which a

diagnosis could be based had not then developed. Probably every ophthalmologist of moderate experience has encountered cases of retinal detachment in which choroidal sarcoma was a possible explanation of the symptom; yet in the absence of other evidence pointing in that direction such a theory was dismissed and in most cases rightly disregarded. Even where there was considerably more reason to suspect sarcoma the failure to make the diagnosis could scarcely reflect on the skill of the surgeon.

Lawford has published a case<sup>1</sup> in which the tumor, seen at the temporal side of the macula, did not cause him to suspect its nature until the patient returned more than three months later. In 1868 Morton<sup>2</sup> reported a case in which iridectomy was done for glaucoma, and two weeks later, there being no relief, the eye was enucleated and found to contain a tumor that occupied one-half the posterior chamber. Again, our competent and careful colleague, the late Dr. W. B. Marple,<sup>3</sup> reported a case in which he had done iridectomy for glaucoma, and within five weeks had enucleated the eye, finding a sarcoma 12 by 14mm. In this case the growth had already extended beyond the globe, so that he did exenteration of the orbit five weeks later.

Such a protracted course as our case pursued is by no means unknown. Zentmayer<sup>4</sup> has recently reported the case of a man, aged forty-nine years, who two years before had noticed a scotoma. Corresponding to the scotoma was a mound-like, steel-gray, somewhat mottled swelling in the fundus. The patient refused enucleation, and disappeared; seven years after the initial symptom he returned with acute secondary glaucoma. The eye had remained quiet, except for one congestive attack. Enucleation revealed mixed-cell melanosarcoma, 10 by 7.5 mm.

Kipp's<sup>5</sup> case was yet more striking. His patient was seen at the age of sixty-four years, with the right retina already detached, but so transparent that two semiglobular vascular

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<sup>1</sup> *Ophthalmic Review*, xxxiv., p. 97.

<sup>2</sup> *Trans. Amer. Ophth. Soc.*, 1868, p. 39.

<sup>3</sup> *Ibid.*, vol. ii., p. 193.

<sup>4</sup> *Trans. College of Physicians of Philadelphia*, xxxvii., p. 429.

<sup>5</sup> *Trans. Amer. Ophth. Soc.*, ix., p. 332.

masses of dark color could be seen through it. She refused enucleation. Sixteen years later she returned because the left eye was becoming blind. A year after she was first seen the right eye had been violently inflamed for many months, and then it became quiet and shrunken. Again enucleation was refused. Six years later, or twenty-two years after it was first advised, the eye was removed for hemorrhage from a fungoid mass that had begun to protrude about one year before. The growth was a melanosarcoma in which spindle cells prevailed.

The case reported by Nettleship,<sup>1</sup> when first seen, was noticed as having a normal fundus, but sixteen months later presented a spot below the macula, with a center of dull grayish-black with definite edges and a pale-gray zone around it, the whole quite as large as the opaque disk. Two years after that the patch still appeared flat and not decidedly larger. Then the patient was not seen for seventeen years, when Richardson Cross examined the case and found a rounded mass, seen with a sph.+10 D. lens, suggestive of sarcoma. Four years later he came to Mr. J. B. Lawford for secondary glaucoma, and the eye was excised, almost twenty-four years after the spot was first noticed.

How often sarcoma starts in the choroid but never develops sufficiently to attract attention and lead to a diagnosis we have no means of knowing. But it is remarkable that several cases of the kind are to be found in the literature. Ginsberg<sup>2</sup> demonstrated to the Berliner Ophthalmologische Gesellschaft, March, 1911, an unpigmented sarcoma, 0.8 by 0.3mm, found in the choroid of a man aged thirty-two dying of nephritis.

At the meeting of this Society in 1911<sup>3</sup> Fuchs demonstrated three such cases, the largest of which was 2.25 by 1 by 0.52mm in size, that were discovered, one *post mortem* in the eye of a patient dying of cysticercus of the fourth ventricle, and the others in eyes enucleated, one for glaucoma, the other for corneal suppurative. When we consider the compara-

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<sup>1</sup> *Royal Society of Medicine*, vi., Section on Ophthal., p. 1.

<sup>2</sup> *Centralb. f. praktische Augenheilk.*, 1911, p. 106.

<sup>3</sup> *Trans. Amer. Ophth. Soc.*, xii., p. 787.

tively small number of eyes submitted to careful anatomic investigation, the number of three choroidal sarcomata thus discovered by one observer seems strikingly large.

The peculiarity of the present case, which it shares with those of Nettleship and Zentmayer, is its long period of gradual development without any interruption by violent inflammation, followed by shrinking of the eyeball. Most of the prolonged cases give a history of general ocular inflammation followed by shrinking, as in Kipp's case, and there are others, like the case reported by Roy,<sup>1</sup> in which it is probable that the sarcoma has developed in an eye previously diseased.

This matter of the early recognition and study of choroidal sarcoma through the period of development is of great importance, as stated by R. Foster Moore<sup>2</sup>: "It is clearly of the greatest importance not to remove an eye in which the only flaw is an innocent stationary growth which causes no symptom and no defect; on the other hand, it is imperative to remove at the earliest possible moment an eyeball containing a growth which is a menace to the patient's life."

The course of these protracted cases indicates that within the eye influences are exerted which may retard or wholly check the development of such growths. One of these is probably the influence on cell life of intraocular pressure. The effect of abnormally high intraocular pressure is fairly well known through the changes produced in previously normal tissues by glaucoma. It is rather probable that the normal intraocular pressure exerts some such influence unfavorable to the cell life of a new-growth.

However, another factor must be considered. Every invasion of the human organism, or development within it of adverse forces, is met with some sort of effective resistance. What forms this resistance takes in the case of sarcoma we do not know. But some of the pathologic phenomena associated with sarcoma are manifestations of that resistance. In the present case two sets of facts are to be considered from this point of view. When the patient was seen February 26, 1915,

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<sup>1</sup> *Trans. American Ophth. Soc.*, xii., p. 188.

<sup>2</sup> *Royal London Ophthalmic Hospital Reports*, xix., p. 424.

the inflammatory reaction present, following exposure to cold, was something more than a mere exacerbation of glaucoma. And, again, when he came to have the eye enucleated, he presented a low-grade panophthalmitis with severe orbital cellulitis. The ring infiltration of the cornea, closely resembling ring abscess, was a striking symptom. The adhesions found during enucleation indicated similar extensions of inflammation from the globe on former occasions.

Such attacks have also been recorded as the most striking feature of other cases of choroidal sarcoma. Veasey<sup>1</sup> records a case in which the eyeball, enucleated in an attack of marked orbital cellulitis, "was found to contain a large lobulated dark growth occupying the greater part of the posterior half of the globe." Myashita<sup>2</sup> reports two cases. In one attention was first attracted to the eye by an attack of "tenonitis." There was floating detachment of the retina that disappeared after scleral puncture. Two years after the first there was a second attack of "tenonitis." Shortly after this, acute glaucoma set in and the eyeball was removed. The sclera was adherent to surrounding tissue, and flat, spindle-cell sarcoma of the choroid was found. In his second case conjunctival ecchymosis set in after vision had been affected for six months. The eye was slightly prominent and its movements were painful. An area of altered choroid was discovered. Six months later the "tenonitis" recurred, and the choroidal patch showed more the appearance of a tumor. The eye was excised, and a spindle-cell sarcoma found in the outer layers of the choroid.

Myashita quite logically associates his cases with the numerous cases of violent inflammation and shrunken globe, and of sympathetic ophthalmia associated with choroidal sarcoma. He lays stress on the harmful products of tissue necrosis as a cause of such inflammation, but it seems possible that the forces in question are operative from the beginning of the growth of the sarcoma. In Myashita's cases the orbital inflammation came early. In our case the most striking thing about the specimen was that it stained so

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<sup>1</sup> *Annals of Ophthalmology*, xx., p. 83.

<sup>2</sup> *Klinische Monatsbl. f. Augenheilk.*, March, 1911, p. 288.

badly, not only tumor-cells and exudates, but even the tissues normally present. Some influence had profoundly interfered with the nutrition of every cell in the eyeball. Coats, commenting on Myashita's paper,<sup>1</sup> calls attention to these "cases in which not only the tumor itself, but all other intraocular structures are necrotic." In reporting on a very small sarcoma of the choroid removed by Buller,<sup>2</sup> Adami says, with reference to certain spindle-shaped collections of pigment: "Not having been able to recognize the nucleus of any of these masses, it is not possible for me to state with absolute certainty that these are cells." The determination as to whether degenerative changes such as are here discussed usually set in before the intraocular tension is notably increased would help to throw light upon their causation.

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<sup>1</sup> *Ophthalm. Review*, xxx., p. 310.

<sup>2</sup> *Trans. Amer. Ophth. Soc.*, vii., p. 378.



REPORT OF A TUMOR OF THE FRONTAL LOBE  
OF THE BRAIN WITH OCULAR SYMPTOMS.  
AUTOPSY.<sup>1</sup>

BY DR. ARNOLD KNAPP, NEW YORK.

*(With two figures in the text and three illustrations on Text-Plates VI.-VII.)*

THE following case of tumor of the frontal lobe is of interest on account of the form of visual-field defect and the cystic protrusion of the floor of the third ventricle.

S. H. V., aged thirty-six years, was first seen on September 11, 1916, on account of failing sight and pain about the left eye which had existed for six months. He had had an attack of grip last winter and has been treated for neuritis. The general health has been good, except that he has been rather irritable, with some mental aberrations and attacks of partial unconsciousness. The patient has been an excessive smoker and a moderate user of alcohol. The Wassermann test was 4+.

Examination of the eyes: V. R. E. = 20/50; L. E. = 2/200, excentric. The pupils contract promptly and then immediately dilate again. The optic disks are atrophic, left more than right, with the characteristics of a descending atrophy. The fields show a central scotoma in both eyes, together with peripheric contraction in the left (Fig. 1). Examination of the nose reveals a deflected septum and left posterior ethmoidal and sphenoidal disease.

The septum was straightened, the left posterior nasal cavities curetted, and some polypoid tissue was removed. This was followed by improved breathing, but no change either in vision or in the headache.

The patient was seen again on March 9, 1917, by the courtesy of Dr. C. G. Coakley. He had been treated with mercurial inunctions, potassium iodid, and strychnin. Vision was reduced to R. E., 2/200; L. E., M. h. in lower field.

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<sup>1</sup> Read at meeting of Am. Ophth. Soc., Hot Springs, Va., 1917.

Pupils moderately dilated, react to light. Field reduced (Fig. 2). The headache had not abated. Patient apathetic, mentally slow, and there is numbness of the right side of the face.

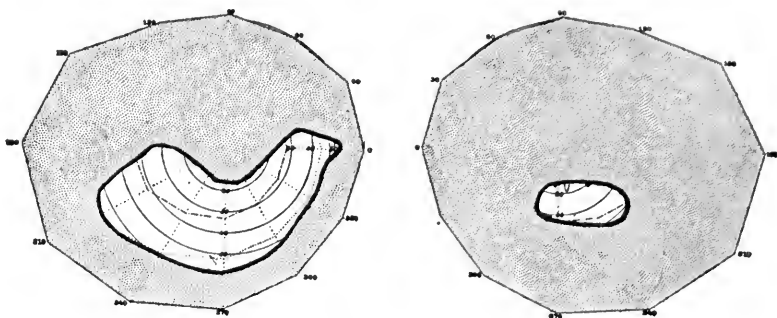


FIG. 1

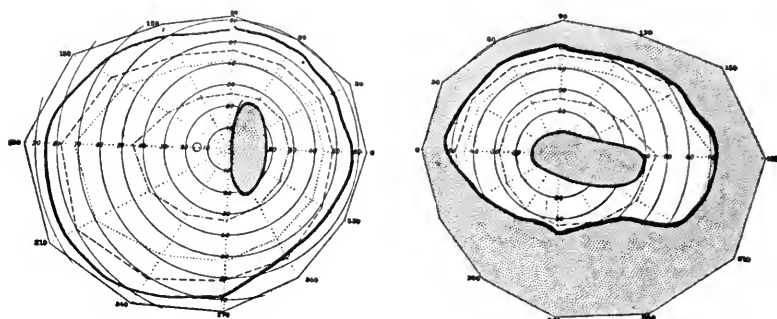


FIG. 2

An X-ray examination was made by Dr. E. W. Caldwell (Fig. 3), who reported that there was external bone absorption in the sella turcica and in the anterior fossa, just anterior to the sella, and above the posterior ethmoidal cells. The cortex of the sella is so completely absorbed that there is no shadow. This absorption suggests pressure by an unusual mass lying in the sellar region, partly in the anterior and partly in the middle fossæ.

He was admitted to the Presbyterian Hospital on Dr. A. V. S. Lambert's service for an exploratory operation. An osteoplastic flap was reflected in the right temporal fossa; general oozing and diploic bleeding were so great that the operation had to be interrupted, and three hours later the patient died.

At autopsy a large tumor measuring 2 by 2½ inches was found in the right frontal lobe. It was an irregular, round

ILLUSTRATING DR. KNAPP'S ARTICLE ON "FRONTAL LOBE TUMOR."

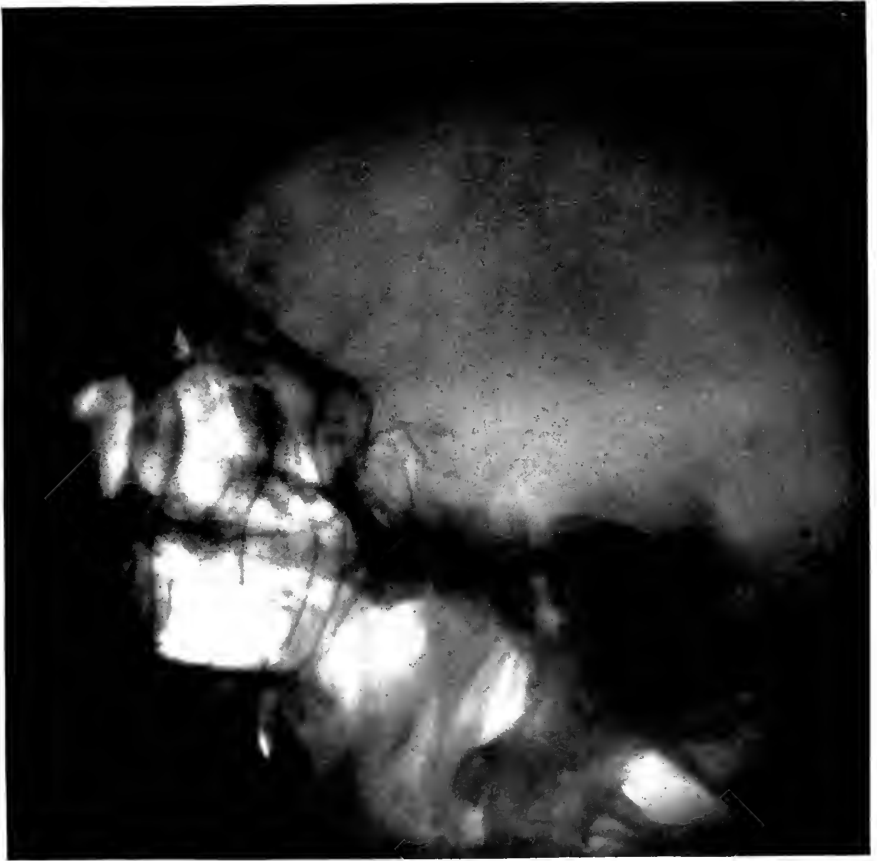


FIG. 3.—X-ray showing excavation and bone rarefaction in ant. and mid. cranial fossae. Deformation of ant. clinoid processes.



mass springing from the dura on the convexity and from the adjoining part of the falx. The tumor was sharply defined and of a very firm consistence, being readily shelled out, and consequently would have been most operable. The microscopic examination showed it to be an endothelioma. The right frontal lobe was greatly enlarged at the expense of the left, so that the median line ran obliquely in the left half of the skull; the cortex of the right lobe was very much reduced in size, even to the naked eye. The bone covering the sphenoidal and ethmoidal cells was very thin. The optic nerves were distinctly flattened. Posterior to the chiasm there was a rounded, bulging mass, a distended tuber cinereum, which had crowded the optic tracts apart and was hiding the mammillary bodies. The wall of this distended area was so thin as to be translucent. The dilatation of the third ventricle was limited to its lowest part, forming a sacculated diverticulum. The hypophysis was the size of a small lima-bean and was displaced backward. The dural covering to the pituitary fossa was intact, and the infundibular opening was not enlarged.

The brain, in brief, showed a frontal tumor in an enormously enlarged right lobe which had produced a sacculated distention of the third ventricle and pressure atrophy of the underlying bone in the anterior and middle cranial fossæ.

The bilateral central scotoma and the excavation of structures anterior to the anterior clinoid processes, as seen in the X-ray, suggested a definite localization of the tumor at the base of the frontal lobe, but the autopsy showed that the tumor was situated at the convexity of the right frontal lobe, next to the falx.

Tumors situated at the base of the frontal lobe are known to produce optic neuritis, optic atrophy, paralysis of the ocular muscles, and exophthalmos, while pressure farther back upon the chiasm or tract causes hemianopsia. Martin<sup>1</sup> collected 61 cases of tumor of the frontal lobe and found in 31 pronounced optic neuritis, in 10 no change, in 3 unilateral optic neuritis, in 7 optic neuritis occurring late in the course of the disease, and in 10 optic atrophy. In 8 cases the optic neuritis was more marked on the side of the tumor, and in 2 it was more marked on the opposite side. The occurrence of unilateral choked disk in brain tumor confirms the basal localization of the lesion.<sup>2</sup> The combination of one-sided

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<sup>1</sup> *Lancet*, July 10, 1897.

<sup>2</sup> Uhthoff: Bowman Lecture, *Trans. Ophth. Soc., U. K.*, 1914.

marked visual disturbance or amaurosis with optic atrophy in association with choked disk on the other side indicates a tumor anterior to the chiasm, which has compressed one optic nerve, causing atrophy and a choked disk of the second eye. The choked disk is prevented on the affected side by the tumor shutting off the communication with the inter-vaginal space of the optic nerve. Schultz-Zehden<sup>1</sup> reported a case in which a tumor starting from the lower surface of the midbrain had invaded the floor of the frontal lobe and penetrated the right lateral ventricle. The right frontal lobe was enormously enlarged. The right optic nerve in front of the chiasm was completely crushed by the tumor and transformed into tumor tissue. There was an optic atrophy upon that side, with blindness, and the other eye presented the picture of a choked disk. Paton<sup>2</sup> has drawn attention to the fact that a frontal tumor may definitely press upon one optic nerve with a central scotoma in the corresponding eye and primary optic atrophy; in the other eye the sight is unaffected, and ophthalmoscopically there is a fairly intense optic neuritis. Paton states that this class of case, in which there is pressure on one optic nerve with optic neuritis in the other eye, is not uncommon, for during the last few years a number of cases of this kind have come to his attention at the Queen's Square Hospital. Three or four cases were recorded in which blindness developed without disk signs of pressure atrophy, and then atrophy developed in five to six weeks.

In this country Foster Kennedy<sup>3</sup> has described this same association of symptoms of a retrobulbar neuritis on the side of the tumor and papilloedema on the opposite side.

In the case reported in this paper the bilateral central scotoma, to my mind, is an evidence of pressure exerted on both optic nerves. The history of excessive tobacco indulgence and the complicating disease of the left posterior nasal cells cannot explain the optic-nerve changes. That this pressure was considerable is shown by the excavation of the sphenoidal and ethmoidal region, as shown by the X-ray.

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<sup>1</sup> *Klinische Monatsbl. f. Augenheilk.*, xliii., pt. ii., p. 153.

<sup>2</sup> *Trans. Ophth. Society, U. K.*, p. 133, 1910. *Brain*, 1909, p. 68.

<sup>3</sup> *Amer. Jour. Med. Sci.*, 1911.

ILLUSTRATING DR. KNAPP'S ARTICLE ON "FRONTAL LOBE TUMOR."



FIG. 4



FIG. 5.—Base of brain showing protrusion of floor of III. ventricle.





The cystic distention of the floor of the third ventricle, as seen in this case, is a frequent finding in internal hydrocephalus. Its presence in a tumor of the convexity of the frontal lobe is interesting. The pressure which it exerted was shown by the X-ray to be sufficient to deform the sella, displacing the normal hypophysis backward. It cannot be stated whether pressure enough was exerted on the tracts and chiasm to cause visual changes in this case on account of the severity of the process in the anterior cranial fossa.

It is known that internal hydrocephalus can cause excavation and pressure-atrophy of the sella turcica from cystic dilatation of the floor of the third ventricle. The chiasm which projects into the anterior wall of the third ventricle is compressed and blindness results. Dyspituitary symptoms have also been produced, and supposedly hypophyseal tumors have been operated upon, but the third ventricle was opened instead. As the posterior part of the chiasm projects into the third ventricle, pressure in hydrocephalus may first be exerted upon this part of the chiasm. It is, therefore, possible that the first change in the visual field is a central bitemporal hemianopic scotoma. This can then lead to a central scotoma or to the development of a bitemporal hemianopsia. Bitemporal hemianopsia as a chiasmal lesion from a distended third ventricle is, however, unusual.<sup>1</sup>

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<sup>1</sup> Henschen: *Berl. klin. Wochenschr.*, 1897, p. 1061. Uhthoff: Bowman Lecture, *Trans. Ophth. Soc., U. K.*, 1914.

REPORT ON THE PROGRESS OF OPHTHALMOLOGY.  
GERMAN REPORT FOR THE SECOND  
QUARTER OF 1916.

By H. KOELLNER, Berlin; W. KRAUSS, Marburg; R. KÜMMELL, Erlangen;  
W. LOEHLEIN, Greifswald; H. MEYER, Brandenburg; W. NICOLAI,  
Berlin; H. PAGENstecher, Strassburg; K. WESSELY, Würzburg;  
and M. WOLFRUM, Leipsic, with the Assistance of Drs. ALLING, New  
Haven; CALDERARO, Rome; CAUSÉ, Mayence; CURRAN, Kansas City;  
DANIS, Brussels; GILBERT, Munich; GROENHOLM, Helsingfors; v.  
POPPE, Petrograd; TREUTLER, Dresden; and VISSER, Amsterdam.

Edited by MATTHIAS LANCKTON FOSTER, M.D., F.A.C.S.,  
New Rochelle, N. Y.

(Concluded.)

XI.—LACRIMAL PASSAGES.

41. SALUS, R. Purulent inflammation of the conjunctiva and lacrimal  
sac caused by the micrococcus catarrhalis. *Klinische Monatsblätter f.*  
*Augenheilkunde*, 1916, i., p. 238.

42. V. SZILY, A. The pathology of the lacrimal passages as shown by  
roentgenography. *Ophthal. Gesellschaft in Heidelberg*, July 31 and Aug. 1.

SALUS (41, Purulent inflammation of the conjunctiva and  
lacrimal sac caused by the micrococcus catarrhalis) saw an  
inflammation in a child a week old which closely resembled  
at first a very severe gonorrheal infection, with intact corneæ.  
At the same time there was a marked swelling of the region of  
the lacrimal sac, pressure upon which caused the expression  
of masses of yellowish white pus from the puncta. Microscopic  
examination revealed typical gram-negative cocci, mostly intra-  
cellular and lying in nests, of the size of gonococci, but the course  
under treatment with recovery in four days contraindicated  
the diagnosis of gonorrheal ophthalmia. The microorganisms  
were proved by culture to be micrococci catarrhalis.

VON SZILY (42, **Pathology of the lacrimal passages as shown by roentgenography**) has investigated the normal and pathological conditions of the lacrimal passages by means of roentgenographs after previous injection of a suspension of very finely pulverized thorium oxidate in fluid paraffine. These pictures show the normal passages, as well as physiological variations, which may give rise to notable changes. Pathological conditions present many forms, although certain types constantly recur. These principal types are two. In the first group the changes start from the so-called isthmus ductus lacrimalis. A contraction takes place beneath the sac with a comparatively broad duct, which later becomes complete. In another large portion of the cases there is a gradually progressive disease and contraction of the duct from below. In both cases large ectasiæ and diverticuli of the sac may appear as time goes on, of which the roentgenograph first gives us a correct conception. He showed also pictures of tuberculosis of the lacrimal passages, of valvular occlusion in which abundant residual contents always remain after the tensely filled sac has been emptied by pressure, of acromegaly, of congenital atresia of the nasal opening, and of battle wounds of the lacrimal passages. Roentgenography promises to be of special importance in determining the benefit obtained by probing, as well as in aiding in the choice of the various operative procedures. K.

## XII.—ORBITS, EXOPHTHALMOS, ACCESSORY SINUSES.

43. AUGSTEIN, H. **Bilateral pulsating exophthalmos caused by a bullet wound.** *Klinische Monatsblätter f. Augenheilkunde*, 1916, i., p. 484.
44. BIRCH-HIRSCHFELD. **Three cases of optic neuritis in association with empyema of the ethmoid.** *Med. Klin.*, 1916, No. 16, p. 427.
45. CORDS. **The causes of death after wounds of the orbit, and their prevention.** *Ophthal. Gesellschaft in Heidelberg*, July 31 and Aug. 1, 1916.
46. JICKELI, C. **A case of traumatic enophthalmos.** *Klin. Monatsbl. f. Augenheilkunde*, 1916, i., p. 247.
47. JICKELI, C. **Two cases of acute inflammatory exophthalmos.** *Wissensch. Vortragsabend der Militäerärzte zu Nagyszeben*, Feb. 5, 1916.

The disease in JICKELI'S (47, **Acute inflammatory exophthalmos**) set in with symptoms resembling those of influenza. The exophthalmos amounted to 4mm and was accompanied by diplopia. The accessory sinuses and the conditions shown

by the X-rays were normal. All symptoms disappeared in a week under conservative treatment. Apparently these were cases of influenza metastasis.

K.

JICKELI'S (46, **Traumatic enophthalmos**) patient, eighteen years old, was struck on the left temple and brow by a brick which had fallen a distance of two meters. Some days later he noticed that his left eye had become smaller and that he saw double, although the eyeball was uninjured and its vision perfect. Examination of the accessory sinuses and with the X-rays revealed nothing wrong. Consequently the immediate onset of enophthalmos and diplopia, as well as the larger left pupil, the lessened power of accommodation, the narrowing of the palpebral fissure, the continued contraction of the pupil in the dark, and the reduction of the corneal and conjunctival reflexes, could be explained only through a change in the suspension apparatus of the globe. The writer thinks that the shock to the orbit caused a sudden stretching, pulling, and partial laceration of the fascia, which enabled the muscles to retract the globe so that each of its movements resulted in disparate retinal images. The dilatation of the left pupil and the weakness of the accommodation indicate a reduction in the tone of the twigs of the left oculomotor nerve within the orbit. The narrowing of the palpebral fissure and the faulty dilatation of the pupil in the dark point to a lesion of the sympathetic, and the reduction of the corneal reflex to an injury of the sensory fibres of the trigeminus. The simplest explanation of all these disturbances seems to be that a moderately large intraorbital hemorrhage took place in the region of the ciliary ganglion, near which the oculomotor and nasociliary nerves pass.

CORDS (45, **Causes of death after wounds of the orbit**) divides his fifty-eight cases into those in which an inflammation of the orbital tissue itself proved fatal, and those in which the wound of an organ more or less near the orbit caused death. The first group is very small. In spite of severity of the wound infection anteriorly, inflammation of the retrobulbar tissue is very rare, and only one case was observed

in which it caused death, a case of bullet wound through the nose and orbit. The treatment consisted of removal of large foreign bodies from the orbit with only careful attempts to extract small ones, drainage, and the greatest possible quiet of the orbital tissues, especially after symptoms of inflammation appeared. Cords observed no tetanus, or gas cellulitis, after wounds of the orbit. Erysipelas occurred only in one case, which did not prove fatal. In the second group he speaks first of infections of the temporal region and pterygomaxillary fossa, and of infections extending out from wounds of the nose and ethmoid, which often lead quickly to thrombosis of the cavernous sinus and later cause empyemata of the accessory sinuses. Of special importance is the frontal sinus, the posterior wall of which is often crushed. He recommends that at the first operative intervention these cavities should be opened freely and given good drainage, or radically extirpated. He considers fissures in the posterior wall of the frontal sinus to be particularly dangerous, and when these are present lays a larger extent of the dura bare. Wound of a large vessel is a rare cause of death; when the carotid artery or the jugular vein is torn at their entrance into the skull, death follows immediately either from hemorrhage or air embolism. He lost one patient from hemorrhage from the internal maxillary artery. Contusion of the brain may cause death, but a direct wound is more to be feared. Tangent shots of the temporal portion of the brain are not rare and may easily be overlooked, as the wound of the brain is apt to be hidden beneath the temporal muscle. Vertical or horizontal tangent wounds of the frontal portion of the brain require the wound to be laid freely bare by a curved incision through the brows or nose, evacuation of the crushed frontal sinus, very careful attention to the wound in the brain, and open treatment. The prognosis of brain wounds through the orbit is very bad. The cause of death in most cases is encephalitis, the next most common is meningitis, and finally abscess of the brain may cause death suddenly, in many cases after the lapse of months.

K.

AUGSTEIN (43, *Bilateral pulsating exophthalmos*) describes a case of this nature caused by a wound in which the muscles

of mastication and the sternomastoid of the left side were completely torn to pieces, the ramus of the lower jaw, the malar bone and the joint destroyed, and the facial nerve completely paralyzed. Later a distinct pulsation of the carotid could be felt in the posterior part of the scar, the principal veins of the upper lids were distended and tortuous, and both eyes protruded, the right 23mm, the left 20mm. No material difference in the exophthalmos when the patient was stooping, or when lying on his back. The intraocular tension was raised. A distinct pulsation, synchronous with the beat of the heart, was felt by the hand placed on the closed lids. Over the entire skull a vesicular murmur could be heard, synchronous with the carotid pulse. Both pulsation and murmur were immediately suppressed by compression of the left carotid. As the patient bore this compression very well, except for a moderate degree of dizziness, the common carotid was ligated, after which both exophthalmos and pulsation subsided. Subsequently fine pigment anomalies appeared in the retinae of both eyes, together with whitish lines of opacity and punctate hemorrhages in the left, although the vision remained normal in both eyes. The exophthalmos may have been caused by rupture in the sinus cavernosus due to explosive action far above the track of the missile, or to the shock of the contusion of the skull.

BIRCH-HIRSCHFELD (44, **Optic neuritis with ethmoiditis**) obtained good results in three cases of optic neuritis through treatment directed to an empyema of the ethmoid. In testing the vision he recommends to add to the methods usually employed the measurement of the blind spot, which is possible only in very intelligent patients. A transient impairment of vision in the third case he demonstrated to be due to an enlargement of the blind spot.

### XIII.—CONJUNCTIVA.

48. BOTTERI. **Vernal catarrh.** *Wiener klin. Wochenschr.*, 1916, No. 15, p. 457.

49. HAAS. **Treatment of gonorrheal conjunctivitis of adults with noviform.** *Wochenschr. f. Ther. u. Hygiene d. Auges*, 1916, No. 29, p. 141.

50. MOHR, M. **Conjunctivitis caused by typhoid bacilli.** *Klinische Monatsblätter f. Augenheilkunde*, 1916, i., p. 523.

51. PASCHEFF. A new conjunctival inflammation—conjunctivitis necroticans infectiosa. *Ophthalm. Gesellsch. in Heidelberg*, July 31 and Aug. 1.
52. POLLNOW. Rare proliferations of vernal catarrh. *Deutsche med. Wochenschrift*, 1916, No. 18, p. 557.
53. ROENNE, HENNING. Treatment of chronic conjunctivitis with optochin. *Klin. Monatsbl. f. Augenheilkunde*, 1916, i., p. 301.
54. SALZMANN, M. Trachoma and gonorrhea. *Arch. f. Dermatologie und Syphilis*, cxx., 1.
55. STARGARDT, K. Ætiology of phlyctenular ophthalmia. *Ophthalm. Gesellschaft in Heidelberg*, July 31 and August 1, 1916.

In cases of chronic conjunctivitis which refused to respond to zinc, silver, and argyrol, ROENNE (53, **Treatment of chronic conjunctivitis with optochin**) obtained a good subjective effect from the instillation of a  $\frac{1}{2}$ ' to  $\frac{1}{4}$ % solution of optochin two or three times a day. He thinks the effect of this remedy, like that of astringents, depends on a biological reaction of the mucous membrane rather than on a direct action on the bacteria.

MOHR (50, **Conjunctivitis caused by typhoid bacilli**) reports a case of this nature caused by the accidental inoculation of an eye. In order not to injure the conjunctival epithelium and permit the deeper entrance of the bacilli, energetic treatment was avoided. The conjunctival sac was irrigated with water and applications made of lukewarm boric-acid solution. The conjunctivitis was mild and quickly passed away. Typhoid bacilli were found in the catarrhal secretion on the second day, but had disappeared at the end of six days.

According to STARGARDT (55, **Ætiology of phlyctenular ophthalmia**), the old eczematous theory must be thrown aside, because it agrees with neither the histopathological facts nor clinical observations. Phlyctenular eye disease is connected only with scrofulosis. As the clinical picture of scrofulosis is composed of two clinical pictures, those of tuberculosis and of the exudative diathesis, it must first be determined to which of these two it belongs. It cannot appertain to the exudative diathesis because phlyctenulæ occur in animals also, so it must be connected with tuberculosis. Such a connection is indicated also by experiences with tuberculin, especially the local reaction after subcutaneous injections. Schumacher and Stargardt obtained positive local reactions in 43%. The

idea that phlyctenulæ are of tuberculo-toxic origin is erroneous. Stargardt's experiments go to show that phlyctenulæ can be formed only when bacilli, or parts of bacilli, are present in the tissue. He likens phlyctenular disease to lichen scrophulorum, and so to cutaneous tuberculide. That this lichen is a true tuberculosis can now be accepted as certain because of positive findings of bacilli and positive results of inoculation, though only in small numbers. As regards phlyctenulæ, only one positive result of inoculation has been obtained, that of L. Mueller, and Stargardt has been the first to find a single case of Much's granular form of tubercle bacillus. The bacillary theory is supported by the findings of Schieck, who repeatedly saw phlyctenulæ in rabbits after the injection of tubercle bacilli into the carotid. A precondition for the development of phlyctenulæ, as for cutaneous tuberculide, is a certain content of antibodies, in consequence of which the bacilli are quickly destroyed, and then, from their remains, phlyctenulæ are formed in the conjunctiva and cornea, lichen nodules and other tuberculides in the skin. The onsets of lichen and other skin tuberculides, and of phlyctenular processes in the eye after acute exanthemata, correspond; in both, according to von Pirquet, the reduction of the content of antibodies plays an important part. The fact that all of the bacilli are not always destroyed gives rise to progressive diseases and to the various pictures that characterize phlyctenular conjunctivitis and keratitis. The frequent localization at the margin of the cornea Stargardt explains through the bad and irregular circulation demonstrated by him in the marginal vascular network. The disease may be of exogenous, as well as of endogenous origin, as indicated experimentally. He believes phlyctenular disease to be a true tuberculosis induced by bacilli in the diseased foci themselves, but very distinct conditions of immunity need to be present, as they occur in benign, not in malignant tuberculosis.

K.

POLLNOW'S (52 **Rare proliferations in vernal catarrh**) nineteen-years-old patient had suffered since his fourteenth year from eye trouble, which set in in the spring and disappeared with the beginning of cold weather. This trouble became



worse in the field. There were commencing proliferations on the conjunctiva of the right upper lid, while on that of the left were very extensive, cauliflowerlike, polypoid excrescences; both the proliferations and the conjunctiva had a dull bluish color. Excrescences at the limbus were wanting in both eyes. No material improvement was obtained by any kind of treatment, including ablation of parts of the growths. He hopes for better success from radical extirpation of the excrescences followed by a pressure bandage retained in position for a long time.

BOTTERI (48, **Vernal catarrh**) has treated fifty cases of vernal catarrh, four of which were complicated by a congestive catarrh which sometimes completely overshadowed the symptoms of the former disease until after its subsidence. This combination of congestive with vernal catarrh may be explained in various ways: (1) The congestive catarrh may be an initial form of the vernal; (2) the cases may have been examples of mixed infection in which there was a simultaneous onset of the two inflammations; (3) the vernal catarrh may have existed for a long time without causing trouble, and the congestive catarrh have been added later. He mentions one case of familial vernal catarrh; two brothers had suffered from this disease since their fourteenth and seventeenth years respectively, and the only son of one of them had suffered from it yearly since his first year.

Between December, 1914, and August, 1915, PASCHEFF (51, **Conjunctivitis necroticans infectiosa**) observed and examined three cases which resembled each other so closely that he believes them to belong to a unique disease not hitherto described. The commencing symptoms are both general and local. The disease sets in with weakness, chill, loss of appetite, frequently headache, and almost always a rise of temperature, which may reach  $38.7^{\circ}$ , associated with a simultaneous swelling of the preauricular and submaxillary lymphatic glands on the side of the affected eye. Locally there are itching and redness of the conjunctiva, slight swelling of the skin near the ciliary margin, the eyes are opened with difficulty, and there is a little photophobia. The secretion is moderate and threadlike. The conjunctiva of the tarsus and transitional folds is reddened,

greatly infiltrated, and fairly smooth. Soon numerous scattered whitish, dull points and spots form in the inflamed conjunctiva, chiefly in the transitional folds and on the tarsi, varying in size from millet seeds to peppercorns. They vary in form, round or oval, and have irregular, indistinct margins. They appear to be superficial and in many cases occupy symmetrical places in the transitional folds from simple contact. After the elimination of the whitish centers there remain slight deep ulcerations of the conjunctiva which soon disappear and leave no trace. The course of the disease on the conjunctiva lasts from two to three weeks and embraces three periods: (1) The period of necrosis, or formation of the whitish spots; (2) the period of ulceration, or cleansing of the necrotic foci; (3) the period of healing. Only one eye was affected in each of the three cases. The adenitis which accompanies the conjunctivitis lasts longer. Commonly it is confined to the preauricular gland, but may affect the submaxillary, the parotid, and spread to the surrounding tissue. The skin is swollen, glistening, and hot. The suppurating glands were removed in these cases, but the bacteriological and experimental examination of them and of their contents gave negative results. Histologically they presented a thickened capsule with numerous granulations. Experimental studies to determine the cause of this disease revealed its great virulence, and the possibility for the reproduction of the whitish spots in the internal organs, especially the spleen. A microorganism with peculiar morphological and biological properties was isolated. Guinea-pigs inoculated with it die in seven or eight days with the same pathological changes, chiefly in the spleen. The microorganism appears like the coccus and bacillus, attains in culture the size of 0.25 to 3mm, is gram-negative, polymorphous, without cilia and capsule; does not grow in the temperature of the laboratory; does not fluidify Loeffler's serum as it does gelatine; forms no pellicle; clouds bouillon; forms no acid; coagulates milk; gives an acid reaction; has no color in agar; gives a weak fluorescence; does not form indol; produces gas; dies in seven or eight days; its virulence diminishes with time. In agar culture for four or five days it becomes threadlike. It is found everywhere, in the secretions, in the sections, and in culture. These facts favor the hypothesis that this micro-

coccus is the pathogenic agent. Macroscopically the disease is characterized by whitish spots on the human conjunctiva and in the spleen of inoculated animals. Histologically the nodular granulations consist of a proliferation of fixed cells with mitoses, leucocytes, and a wall at the periphery with lymphocytes. Commonly there is a central necrosis, occasionally suppuration in the conjunctiva, almost always in the glands, but no giant cells. The epithelium over the nodules in the conjunctiva is broken through so that the contents may be emptied. In the spleen there are likewise focal proliferations of tissue with a tendency to breaking down and necrosis of their centers, but here also with no giant cells. These changes have hitherto been unknown in human pathology. Prognosis: The spots on the conjunctiva disappear in two or three weeks and leave no traces. The swelling of the glands lasts for months and ends in suppuration. Treatment: Disinfection of the conjunctiva with the usual means. Operation on the swollen glands when they suppurate.

#### K.

Concerning the relations between trachoma and gonorrhea, SALZMANN (54, **Trachoma and gonorrhea**) says that no uniformity of opinion has yet been reached concerning inclusion corpuscles. It is not yet positively determined what importance these conditions have in trachoma and inclusion blennorrhea, and it is still a question whether these two diseases are related or not. The simultaneous occurrence of inclusions and gonococci is too rare to make a connection between the two probable. The clinical courses and the spread of the two diseases differ. An infection with gonorrhea from a perfectly pure inclusion blennorrhea has not yet been observed. The involution forms of the gonococcus show certain resemblances to elements of the inclusion corpuscles, but this is also true of other gram-negative cocci. The virus of trachoma and of inclusion blennorrhea is filterable, that of gonococci is not. While the former are transmissible to monkeys, no one has yet succeeded in an inoculation with young or old gonococci. The inoculation experiment of Herzog on man is not conclusive, because it is not certain that the culture was pure.

Consequently trachoma and gonorrhea cannot be said to be identified.

K.

HAAS (49, **Treatment of gonorrheal conjunctivitis of adults with noviform**) describes a case of acute gonorrheal conjunctivitis in a man twenty-seven years old, in which the cornea was intact, but the œdema of the lids, the chemosis, and secretion of pus were great. Great quantities of gonococci were demonstrated microscopically. The patient came on the third day of the disease and was treated for four days with irrigations of oxycyanide solution and instillations of choleval three times a day. The disease did not advance, but no improvement appeared. On the seventh day of the disease daily applications of a 10% ointment of noviform were begun, which acted so energetically that no trace of the inflammation remained at the end of a week. This brilliant result the writer ascribes wholly to the noviform.

#### XIV.—CORNEA AND SCLERA

56. AUGSTEIN, C. **Specific chemotherapy of ulcus serpens.** *Muenchener medizinische Wochenschrift*, 1916, No. 15, p. 530.

57. AXENFELD. **Bilateral primary progressive parenchymatous calcification of the cornea (dystrophia calcarea).** *Ophthalm. Gesellschaft in Heidelberg*, July 31 and August 1, 1916.

58. ERGLETT. **Correction of keratoconus.** *Muenchener medizinische Wochenschrift*, 1916, No. 17, p. 609.

59. FUCHS, E. **Secondary scleritis and episcleritis.** *Ophthalm. Gesellschaft in Heidelberg*, July 31 and August 1, 1916.

60. OESTERREICHER, L. **A case of implantation cyst after Kuhnt's conjunctival keratoplastic.** *Klin. Monatsbl. f. Augenheilkunde*, 1916, i., p. 157.

61. UHTHOFF, W. **Further clinical and anatomical contributions to keratoconus.** *Ibid.* p. 385.

62. VON WEHDE. **Optochin treatment of ulcus corneæ serpens.** *Diss.*, Rostock, 1916.

UHTHOFF (61, **Keratoconus**) reports two cases in which he was able to examine eyes with keratoconus after enucleation. In the first case the corneal lamellæ became gradually thinner from the periphery towards the center, in the other the cornea, which had a fairly even thickness, had an ectasia in the center

in which the thinness appeared suddenly. In both patients the other eye had a distinct keratoconus with a gradually increasing ectasia and thinning of the substance of the cornea. In the region of the keratoconus Bowman's membrane was in great part absent. Descemet's membrane showed a defect in both cases, which was thought to be secondary to the ectasia, rather than a primary cause of the latter. The changes in the stroma in the central ectasia were, in the first case, an extensive homogeneous hyaline degeneration and a destruction of the corneal corpuscles; in the second case, the central thinning was very considerably due to atrophy and loosening of the corneal tissue, with fissures parallel to the surface, and a slighter degree of homogeneous hyaline degeneration. No brownish ring could be found in the deep layer of epithelium. Both patients had slight tachycardia, one a slight leucocytosis, the other a slight struma, but there was no symptom of disease of any of the glands with internal secretion. Unthoff does not believe that these slight disturbances were connected ætiologically with the keratoconus.

ERGELLET (58, **Keratoconus**) had a patient 16 years old who had suffered from keratoconus for three years. The vision of the right eye was fingers at  $1\frac{1}{2}$  meters, of the left at 1 meter. Correction with a contact glass gave a vision of  $\frac{5}{7}$  to the right eye,  $\frac{5}{7}$ — $\frac{5}{10}$  to the left. The advantages of the contact glass are described and the writer thinks that it is theoretically ideal. The only disadvantage is that not every eye can endure the contact for any length of time. So this patient developed a lesion of the epithelium of her cornea after she had worn the glass for forty-eight hours. After that she was able to wear the glass a few hours two or three times a week without trouble.

AXENFELD (57, **Dystrophia calcarea**) describes a case of a new form of this trouble, which was bilateral, began in youth, and developed gradually and indolently a peculiar circle about the center of the cornea. Otherwise the patient was perfectly well. The opacity was seated beneath Bowman's membrane in the parenchyma of the cornea, where particles of lime gradually settled, with a slow atrophy of the tissue. Treatment was ineffective.

K.

AUGSTEIN (56, **Chemotherapy of ulcus serpens**) speaks energetically in favor of the optochin treatment of the pneumococcus ulcer. It is of the utmost importance to differentiate immediately a diplobacillus from a pneumococcus ulcer, for the writer believes that the former can always be cured by energetic treatment with zinc, and the latter by treatment with optochin. He considers the best way to use optochin is to saturate a bit of sterile cotton with a two per cent. solution, apply it to the ulcer, and leave the eye closed for from five to ten minutes; then instillation of a one half per cent. solution twelve to fifteen times a day, perhaps a couple of times at night. On the following days a reapplication of a two per cent. solution on cotton, according to the severity of the case. He says the results are "staggering." The final result is a more delicate cicatrix than can be obtained by other methods of treatment. He believes that every eye with an ulcus serpens can be saved by this specific treatment if seen in time.

VON WEHDE (62, **Optochin treatment of ulcus serpens**) gives the results of this treatment in the clinic at Rostock. Total number of cases in three years, 38. Optochin used in one per cent. solution, instilled six or eight times a day, with lukewarm applications and scopolamine. In thirty-one the ulcer was brought to a standstill, in seven it was not. He concludes that the results are very good when the ulcer is not too far advanced.

After abscission of the prolapse of iris from a wound of the eye, OESTERREICHER (60, **Implantation cyst after conjunctival keratoplastic**) covered the corneal wound with a conjunctival flap. Six months later he found a tumorlike mass, measuring 10mm by 6mm, attached to the cornea and protruding between the lids when the eye was open. It was not movable, was transparent, and was covered by thin bulbar conjunctiva. On extirpation it was found not to be in communication with the anterior chamber. Such an implantation cyst after this operation has not been observed before, to his knowledge, although they are not so very rare after operations for strabismus and pterygium.

FUCHS (59, **Secondary scleritis and episcleritis**) found in cases of chronic iritis and iridocyclitis, spontaneous, traumatic,

and sympathetic, an infiltration of the tissue of the sclera with lymphocytes and frequently with plasma cells, while polynucleated leucocytes were very scarce. The infiltration may be general or focal, sometimes is in the form of well circumscribed nodes, especially along the vessels. Schlemm's canal and the anterior ciliary vessels are always infiltrated the occlusion of the sinus of the anterior chamber by the root of the iris makes no difference in this respect. Along the posterior openings infiltration was found only in the minority of cases, mainly along the vortex veins, then along the ciliary arteries and nerves. Outside of the sclera, infiltration is always present from the limbus to the insertions of the recti, and is much greater at the limbus than along the anterior openings for vessels. In the posterior zone the infiltration is always far less dense than in the anterior, is never general, but always focal. It is localized in the episcleral tissue and Tenon's capsule; about the blood-vessels frequently as a prolongation of the infiltration about them in the sclera, and little foci of lymphocytes are frequent about the vessels which lie in close relation to the entrance of the optic nerve; about the blood-vessels which lie in the superficial layers of the sclera itself; and at the insertions of the ocular muscles. The infiltration in the posterior segment is always less than that in the anterior. In the latter, the aqueous has immediate access to Schlemm's canal, while the posterior passages drain only the perichoroidal space, which commonly is not much affected by the inflammation. The inflammation extends to the posterior segment of the surface of the globe in chronic cases either through the posterior openings, or, when these are normal, from the anterior segment, from which the irritating tissue fluid passes backwards in Tenon's space. The latter form of extension is the common one in purulent endophthalmitis, in which the surface of the posterior segment shows no sign of inflammation in the greater number of cases. In cases of panophthalmitis which perforate the sclera, a special infiltration takes place at the point where rupture takes place later; in fulminating cases this infiltration is spread over most of the sclera. Thickly packed polynucleated leucocytes lie in the outer layers of the sclera, while in the inner layers the nuclei of the scleral fibres are partially destroyed. Here

another means of transference of the inflammation from within outwards is to be found, by diffusion of toxic substances directly through the sclera with an attraction of polynucleated leucocytes. The process is the same as that which takes place in ring abscess of the cornea. The protrusion of the eye at the beginning of a panophthalmitis is caused by the saturation of the superficial layers of the sclera and the episcleral tissue with fluid containing fibrin, to which are added infiltration with lymphocytes and later proliferation of connective tissue and a new formation of vessels, which render the sclera adherent to the neighboring tissues.

K.

## XV.—IRIS, PUPILS

63. FUCHS. **Shriveling of the iris.** *Klin. Monatsbl. f. Augenheilkunde*, lvi., p. 145.

64. METZNER and WOELFLIN. **Clinical and experimental studies of paresis of the cervical sympathetic.** *Archiv f. Ophthalmologie*, xci., p. 167.

Although shriveling of the iris, usually with ectropion of the uvea, is not rare in connection with increase of tension, FUCHS (63, **Shriveling of the iris**) has observed it with diminished tension. Three of his reported cases were traumatic, one of them being of crushing of the eye from behind. One case was of detachment of the retina in a soft eye, in another an exudate had formed in the vitreous from some unknown cause. The iris was so shrunken that it was scarcely visible at the margin of the cornea. In the cases in which the process could be observed, this great diminution of the iris took place in from eight to fourteen days. One anatomical examination was possible, which showed that the shortening of the iris was caused by a membrane that filled the sinus of the anterior chamber and spread from there over the anterior surface of the iris. The margin of the pupil was drawn over upon the anterior surface of the iris so as to cause an ectropion uveæ. The tissue of the iris was drawn together but not atrophic. The exudation was confined to the posterior parts of the ciliary processes and the anterior surface of the iris. Because of its thin endothelial covering the anterior surface of the iris is more sensitive to irritating substances than the posterior. The posterior layer of the retinal pigment epithelium was



shoved over the anterior. It is evident that to produce this sort of shriveling of the iris it is necessary to have a moderate degree of inflammation of the anterior surface of the iris and an absence of posterior synechiæ, as when it is fixed in this manner such a shrinkage does not occur. The formation of synechiæ was prevented by a lifting up of the iris from the lens, by the sinking back of the latter induced by a loss of vitreous, or by a detachment of the retina, and by the tension of shrinking masses in the vitreous space.

In order to obtain a closer insight into the various symptoms of paresis of the sympathetic, METZNER and WOELFFLIN (64, **Paresis of the cervical sympathetic**) instituted a series of experiments on rabbits. First the superior cervical ganglion was removed in order to study the question of the depigmentation of the iris. Nothing of this sort could be demonstrated, even by diascleral illumination, although the time allowed for observation was sufficiently long. The depigmentation described by other writers appears to have been due to a trophic disturbance. No essential changes in the fullness of the retinal and conjunctival vessels could be found. Sometimes there was a slight degree of exophthalmos. Differences in the retraction of the nictitating membrane were less marked. On the other hand, a contraction of the pupil of the eye on the side operated on appeared immediately and persisted with one exception. Then the sympathetic below the superior cervical ganglion was resected. No depigmentation appeared in the iris, but the pupil became still more contracted than after removal of the ganglion itself, so this part of the sympathetic must furnish an independent tone. Transient symptoms of paresis can be excited by stretching of the sympathetic, the duration of which varies with the degree of stretching, but may be permanent. A permanent myosis appears after removal of the middle ear, though the vasomotor disturbances retrogress. Similar pupillary trouble may occur in men after inflammation of the middle ear, though only a few cases have been reported.

#### XVI.—SYMPATHETIC OPHTHALMIA

65. SCHIECK. **Prevention of sympathetic ophthalmia in wounds of the eye received in battle.** *Ophthalm. Gesellschaft in Heidelberg*, July 31 and August 1, 1916.

SCHIECK (65, **Prevention of sympathetic ophthalmia**) controverts the idea that we do not become acquainted with sympathetic ophthalmia in the course of this war. Wounds of the eye received in battle form no exception to the rule, and need just as careful attention as wounds received during times of peace. It is only the fact that preventive enucleation is the method which has been adopted by surgeons that has prevented the occurrence of many cases of sympathetic ophthalmia. He reports eight cases that have followed shot wounds. In one of these it followed an opticociliary resection. In three the wounded eye was not removed until after the outbreak of the sympathetic trouble. In the remaining four the wounded eye was enucleated at a time when the other eye was perfectly intact; the enucleations proved not to be absolutely protective, although the cases ran mild courses. A certain percentage of such mishaps has always been observed, and it is to be expected that this percentage would show itself among the extraordinarily large number of preventive enucleations which have been, and still must be, performed in this war.

K.

## XVII.—LENS.

66. BACHSTÉZ. **Encapsulation of several foreign bodies in the lens.** *Klinische Monatsblätter f. Augenheilkunde*, lvi., p. 492.

67. FLEISCHER. **Myotonia atropicans and cataract.** *Ophthalm. Gesellschaft. in Heidelberg*, July 31 and August 1, 1916.

68. FUCHS. **Lymphangioma at the place of a cystoid cicatrix.** *Klinische Monatsblätter f. Augenheilkunde*, lvi., p. 145.

69. SCHEUBE. **The nonoperative treatment of senile cataract.** *Inaug. Diss.*, Jena.

FUCHS (68, **Lymphangioma at the place of a cystoid cicatrix**) found a dilatation of the lymphatics in the swollen conjunctival tissue in an eye which had been enucleated on account of a mild endophthalmitis ten days after an extraction of cataract. These lymphatics passed through the scar left by the operation and spread through the young connective tissue lying on the posterior surface of Descemet's membrane and the stump of iris, and also penetrated into the most posterior layers of the sclera. A number of these vessels went directly through the sclera to the ciliary body, which they permeated. The scleral fibers were not simply pushed aside, but were directly worn

away. The vessels were lined with continuous endothelium. This new growth is to be looked upon anatomically as a lymphangioma.

SCHÉUBE (69, **Nonoperative treatment of senile cataract**) believes that medicamental treatment of senile cataract must be considered ineffective because the best results which have been claimed for it are a partial clearing up of opacities and an improvement of the vision.

BACHSTEZ (66, **Several foreign bodies in the lens**) saw several little foreign bodies in a lens, which had been there for seven years and had caused no irritation aside from quite circumscribed opacities. There were similar opacities in the cornea, and an aperture in a corresponding place in the iris. He thinks it probable that the foreign bodies were of iron.

Myotonia atrophicans is easily to be recognized through the characteristic facies myopathica, caused by an atrophy of the muscles of the face, especially of the orbicularis oculi, with the production of a slight lagophthalmos, and of the orbicularis oris, through a difficulty of speech induced by atrophy of the musculature of the larynx, lips, cheeks, and tongue, and through atrophies in certain other regions, such as of the sternomastoid, or of the small muscles of the hand. Serious disturbances of the general nutrition, atrophy of the genital organs, vasomotor symptoms, and psychic troubles, together with the presenile cataract, which is not uncommon, mark the disease as due to a severe disturbance of an internal secretion. Among the cases of cataract at Tuebingen since 1900, FLEISCHER (67, **Myotonia atrophicans and cataract**) has found fifteen cases, eleven of which were subjected to a neurological examination. It is probable that the actual number present in this material was greater, but this is not certain. The ophthalmologist frequently has the first opportunity to recognize this rarely diagnosed disease, as it is the cataract which first leads the patient to consult a physician in a percentage of the cases which is not small.

K.

#### XVIII.—GLAUCOMA.

70. GERTZ. A case of detachment of the retina after Elliott's trephining. *Ophthalm. Gesellschaft v. Copenhagen*, March 16, 1916.

71. GUNNUSSEN. Tonometry of buphthalmos in a sleeping patient. *Klinische Monatsblätter f. Augenheilkunde*, lvi., p. 428.

72. ISCHREY. A case of primary sarcoma of the orbit with anterior adhesive iridocyclitis (Knies) in the luxated eyeball. *Klin. Monatsbl. f. Augenheilkunde*, lvi., p. 492.

73. KOEPPE. The rôle of the iris pigment in glaucoma. *Heidelberger ophthalmische Gesellschaft*, July 31 and August 1, 1916.

74. SEEFELDER. Concerning the relations of the so-called megalocornea and megalophthalmos to congenital hydrophthalmos. *Klinische Monatsblätter f. Augenheilkunde*, lvi., p. 227.

GERTZ (70, Detachment of the retina after trephining of the sclera) reports the case of a girl 20 years old, who had formerly been operated on for lamellar cataract, on whom Elliott's operation with iridectomy was performed for glaucoma. Some vitreous escaped from the wound. On the next day there was a great increase of tension with hemorrhage into the anterior chamber. The increased tension was reduced by eserine. About sixteen days after the operation the eyeball was soft and a nasal defect appeared in the field of vision, and later the entire retina became detached.

KOEPPE (73, The rôle of the iris pigment in glaucoma) demonstrated a more or less marked destruction of the pigment epithelium of the iris in more than forty cases of acute or chronic primary inflammatory glaucoma. This showed itself through a wandering out of dark brown and free pigment material, partly as very minute, partly as larger particles from the cells into the stroma, so that here and there, especially near the veins and the anterior limiting layer, it appeared to be permeated by them. Typical for this "pigment displacement" is its perfect irregularity with respect to extent, site, and distribution. Pigment dust is also to be found on the surface of the iris, on the corneal endothelium, and within the marginal portions of the cornea, as well as on the anterior and posterior capsules of the lens. In acute glaucoma more or less pigment dust is visible in the anterior chamber. Microscopically with lithium carmine stain and with the oil immersion, a very fine, dark brown free pigment material is to be seen, partly as little balls, partly as true dust, free in the stroma of the iris and in part of the ciliary body, especially in the lymph spaces and adventitial lymph sheaths of the vessels or

veins, particularly toward the anterior limiting layer. In more advanced cases of glaucoma may be seen separate light brown scales of pigment, likewise sometimes the result of the secondary destruction of the stroma pigment free in the stroma of the iris. All of these things can be observed more or less in healthy eyes, usually in both. The suspicion of the specificity of the pigment displacement for glaucoma is now confirmed by five cases, in which after an early diagnosis of glaucoma, or, as the author calls it, of preglaucoma, when there were no recognizable symptoms of the disease, glaucoma appeared and could be diagnosed from clinical manifestations. Because of this Koeppel believes it may be assumed that the pigment displacement has an ætiological significance perhaps for glaucoma simplex, and propounds the following theory: In consequence of a trophic disturbance, or a congenital weakness, a slow but irresistible morphological and biological cell destruction begins in the hitherto intact pigment epithelium, as well as in its derivatives, in the iris, and, in part, in the ciliary body. Just as powdered iron in Erdmann's experiment occludes the anterior outflow passages, so the free pigment globules, and dust which has undergone a certain chemical change and therefore become toxic, may possibly occlude the lymph passages and spaces, particularly the perivascular ones of the iris, chiefly at their openings. In addition this may lead perhaps to a contraction and gradual obliteration of the lumen and walls with the result that the stroma of the iris is affected by a slowly advancing atrophy. In this way a condition may be produced which is nearly related to, if not identical with, the glaucomatous predisposition. Thus is brought about a gradual decompensation of the circulation of the lymph, lymphatic engorgement, and the picture of lymphostatic glaucoma. Through involvement of the walls of the smallest capillaries and veins this may develop into hæmostatic glaucoma. If the blood pressure is increased suddenly through the sympathetic, the result is an acute attack of glaucoma. In the opinion of the author the absorptive action of the iris must be considered together with the sinus of the anterior chamber as of the first importance to the outflow from the anterior portion of the globe, as already claimed by Hamburger, the more so as they are often found com-

pletely occluded in glaucoma when the compensation of the sinus of the anterior chamber still exists.

K.

ISCHREYT (72, **Primary sarcoma of the orbit with anterior adhesive iridocyclitis**) had the opportunity to see a peculiar case of orbital sarcoma, which first came under observation as the result of a slight wound. There was much exophthalmos and the symptoms were steadily increasing. Incision into the orbit evacuated no pus. A transient pulsation appeared in the exophthalmic eyeball. Exenteration of the orbit revealed a necrotic sarcoma with a large cyst. A very interesting condition found in the enucleated eye, in addition to degeneration of various layers of the retina, was a close attachment of the root of the iris to the cornea and sclera at the sinus of the anterior chamber, so close that in places the line of demarcation could not be recognized. There was no special intermediate tissue. Some endothelial threads extended across the newly formed sinus. In spite of this fact the tension was not increased, but diminished. In an attempt to explain why glaucoma did not appear, it is suggested that the serious disturbances of the blood supply and nutrition prevented an increase of tension. Among 24,000 cases Ischreyt has seen nineteen with tumors of the orbit, of which eight were primary and eleven were extensions from tumors in neighboring parts.

GUNNUFSEN (71, **Tonometry of buphthalmos in a sleeping patient**) found that the tension during sleep was always materially lowered, the more so the higher the original tension. Three factors come into play for this reduction of tension: the reduction of the blood pressure; the myosis during sleep, which is particularly marked in children; and the decrease of secretion during sleep.

Opinions are divided as to whether megalocornea or megalophthalmos is to be looked upon as a giant growth, or as one of the symptoms of hydrophthalmos. SEEFELDER (74, **Megalocornea and megalophthalmos**) inclined formerly to the latter view, but has concluded from a case that came under his observation that the enlargement of both eyes may be due

not to a process of stretching, but to an abnormal degree of growth. The patient in question was sent to him for examination on account of large eyes when the former came to enter the military service. The diameter of the cornea was 14.5mm, the radius 7.2 vertically, 7.6 horizontally. The conditions which led Seefelder to exclude hydrophthalmos were: The absence of any corneal opacities, of any fissures in Descemet's membrane, and of any broadening of the limbus, the sharpness of the corneoscleral margin, the normal appearance of the sclera, even in the region of the anterior chamber, which was 8mm deep, the posterior part of which was bounded by the sclera, the absence of a glaucomatous cup, and of any disturbance of function, the regular astigmatism, the absence of increased tension, and the correspondence of the measurements in the two eyes. He suggests the name gigantophthalmos for such cases as this.

#### XIX. RETINA AND OPTIC NERVE.

75. AXENFELD. Intraocular irradiation therapy. *Ophthalm. Gesellschaft in Heidelberg*, July 31 and Aug. 1, 1916.

76. HIPPEL, E. V. Signification of choked disk in shot wounds of the brain. *Ibid.*

77. HIRSCHBERG. Central recurrent retinitis in syphilitics. *Zentralbl. f. prakt. Augenheilkunde*, March-April, 1916.

78. IGRSHEIMER. A new way to recognize morbid processes in the optic tract. *Ophthalmische Gesellschaft in Heidelberg*, July 31 and August 1, 1916.

79. LODBERG. A case of chorioretinitis juxtapapillaris (Edw. Jensen) with stellate figure in the macula. *Klin. Monatsblaetter f. Augenheilkunde*, February-March, 1916.

80. MUELLER. Intracranial increase of pressure with choked disk. *Muenchener medizinische Wochenschrift*, No. 20.

81. ROENNE. A case of acute retrobulbar neuritis with wandering defect in the visual field. *Klin. Monatsbl. f. Augenheilk.*, February-March.

HIRSCHBERG (77, Central recurrent retinitis in syphilitics) reports a case of retinitis which he has followed for twenty-seven years. It recurred frequently and responded well to antisyphilitic treatment. After one attack a parafoveal focus remained, in the neighborhood of which during recurrent attacks fresh, bluish infiltrations appeared in the retina, which suggested a partial blocking of the blood by the old focus. The final result was a scotoma with full central vision.

LODBERG'S (79, **Chorioretinitis juxtapapillaris**) case was characterized by a stellate figure in the macula although no nephritis was present. The stellate figure underwent involution and the vision rose from  $\frac{1}{18}$  to  $\frac{5}{6}$ .

AXENFELD (75, **Intraocular irradiation therapy**) stated that the case reported by him and others in 1915 of bilateral glioma retinae had been kept under observation and that the retrogression of the three gliomata induced by irradiation with hard gamma rays had continued until the largest tumor had completely disappeared, leaving only an evidently dead preretinal trace. An expected cataract developed in this eye late in the fall of 1915, and was extracted in the summer of 1916. The restoration of vision to the child, now three years old, was delayed, as the six months' blindness during the formation of the cataract had induced a condition like that of amaurosis after blepharospasm, which passed away after about three weeks. No recurrence of the growth had taken place meanwhile. How far the function of the retina is impaired must be determined later. Otherwise the child is well developed physically and intellectually. The heavy doses of rays administered are plainly not to be borne by the growing lens, but this is not a contraindication to irradiation therapy for glioma. It is only in the second eye, and only when the vision has not been too greatly impaired, that irradiation treatment should take the place of enucleation in glioma.

K.

MUELLER (80, **Increase of intracranial pressure with choked disk**) recommends as an operative measure for choked disk due to cerebral affections the trephining of the sheath of the optic nerve. After Kroenlein's operation a piece of the sheath measuring four by eight millimeters is removed. Choked disk undergoes involution not only upon the same side, but in many cases on the other also.

VON HIPPEL (76, **Choked disk in shot wounds of the brain**) emphasizes the need of a differential diagnosis between choked disk and papillitis in every case, though this is not always attainable. Choked disk is very frequent soon after shot



wounds of the skull. Often it quickly undergoes involution, either spontaneously or under the influence of proper surgical treatment of the wound. Conditions that favor increased intracranial pressure are present after shot wounds of the skull, whether the dura has been wounded or not. Most of the swellings of the papilla seen early after such wounds, which have been called by various names in literature, are true choked disks. The swellings of the papilla that appear late are mostly due to abscess of the brain, meningitis, or a cyst, and may be either choked disk or papillitis. The prognosis of choked disk after wounds of the skull is good in so far as that if the patient lives the swelling of the papilla is likely to go down, but whether the choked disk is or is not a bad sign cannot be stated with certainty. Choked disk alone in the early stage calls for no special treatment, least of all for an operation, but as in most cases it is only one symptom out of many, and not infrequently the general condition necessitates an operation, opportunity is often afforded in gunshot wounds of the brain to see the prompt retrogression of a choked disk after an operation that reduces the pressure. A swelling of the papilla appearing in the late stage shows with certainty that a cure was only apparent and forms an imperative indication for surgical intervention.

K.

IGERSHEIMER (78, **A new way to recognize morbid processes in the optic tract**) ascertained that every interruption of conductivity, or serious disturbance of a bundle of fibers in the optic nerve manifests itself externally as a scotoma extending outwards from the blind spot. Hence it is possible to demonstrate with great exactness even very slight troubles of both the peripheral and the central bundles in the path of conduction. This is very often of considerable importance not only for the early diagnosis, but also for the prognosis and treatment. The essential feature of the method is perimetry vertical to the course of the nerve fibers in the retina. He uses a disk two meters in diameter with a radius of curvature of one meter. It has been demonstrated anatomically that most of the decussated and non-decussated fibers describe concentric arches from the blind spot to a point of fixation and then

bend off to the periphery. This furnishes an easy explanation of the occurrence of a ring scotoma in affections of the optic nerve. The isolation of the papillomacular bundle pathologically is made doubtful, as Igersheimer almost always could demonstrate, in addition to the defect of the central fibers, one of the peripheral in cases of alcohol-tobacco amblyopia, and retrobulbar neuritis caused by affections of the accessory sinuses. It was shown how the new method is able to reveal early affections of the optic nerve in glaucoma, troubles of the anterior segment of the globe, such as iritis, contusions, and perforating wounds, as well as in affections of the orbit, and in multiple sclerosis, tabes, and nephritis. Depending on the same principle disturbances at the chiasm and in the central optic tract are demonstrable at a much earlier period than heretofore.

K.

ROENNE'S (81, **Retrobulbar neuritis with wandering defect in the visual field**) case began with loss of the nasal lower quadrant, which was replaced by a relative paracentral scotoma to the nasal side of the center. This defect gradually moved into the upper nasal quadrant of the visual field, where it grew less marked and finally ended with a distinct, though not strongly accentuated, hemianopic boundary line in the upper nasal quadrant, before it passed over to complete recovery.

#### XX.—ACCIDENTS, WOUNDS, FOREIGN BODIES, PARASITES.

82. HERTEL: **Wounds of the eye by foreign bodies in war.** *Ophthalmische Gesellschaft in Heidelberg*, July 31 and August 1, 1916.

83. LOWENSTEIN. **Wounds of the eye in war in the mountains.** *Ibid.*

84. OLOFF. **Psychogenous injuries of the eyes in war.** *Ibid.*

85. PINCUS. **Clinical observations of shot wounds of the occiput.** *Ibid.*

86. UHTHOFF. **Injuries of the central visual tracts and centers in wounds of the skull.** *Ibid.*

Wounds of the eye form two per cent. of the entire number of wounds in battles on plains and wooded hills, according to LOWENSTEIN (83, **Wounds of the eye in mountain war**), as observed by him in Serbia and Carpathia. But in the Alps he has found them, after twelve months' service, to amount to eight per cent. The great majority of these wounds are

caused by bits of stone, seventy-two out of ninety-eight perforating wounds, thirty-five being of both eyes. Next to the bits of stone come splinters of metal, about equally divided between the magnetic and the non-magnetic. In most cases the foreground of the clinical picture is occupied not by the wound of the eye, but by the simultaneous lesion of the skull. The bits of stone driven into the face caused suppuration for the most part and precluded the possibility of an aseptic operation on the eye. Very often it was hard to differentiate between a contusion and a perforating wound when the anterior chamber was filled with blood and the wound of perforation was minute. Contusion of the cornea gave the writer a clinical picture hitherto unknown to him, which was much more common—observed fourteen times—than Vossius's ring opacity of the lens, of which only three were seen. *Commotio retinae* and ruptures of the choroid were frequently to be seen after contusions with stones. Bits of stone become encapsulated in all the tissues of the eye, for the most part without reaction. Five cases of bits of stone in the iris, which could not be extracted immediately on account of the suppuration in the face, remained without reaction for eight weeks. Of twenty-one wounds of the lens by bits of stone, seventeen showed an opacity of the posterior cortex immediately after the lesion, which ended in all seventeen in total opacification. In four the opacity, which was of no less extent, was localized in the anterior cortex, and none of these showed any progression whatever. Only seventeen blind eyes had to be removed out of a total of 133 perforating wounds. As he had not met with sympathetic ophthalmia in his cases he employed conservative treatment so far as possible for wounds of the eye incurred in mountain war.

K.

HERTEL (82, **Wounds of the eye**) found bits of steel in sixty per cent. of 242 cases of intraocular foreign bodies, the remainder consisting of copper, brass, powder, stone, straw, wood, and glass. The wounds presented almost uniformly such a picture as is produced by blasting, or explosions, multiple lesions in the face, both eyes struck in twenty-one per cent., and not rarely several splinters in one eye. The force of impact usually was

very great, as shown by the great hemorrhages within the eye, the penetration of particles as minute as  $\frac{2}{10}$  mg into the posterior segment of the eye, the frequent occurrence of wounds of entrance through the closed lids often with tearing of pieces of bone from the wall of the orbit, and the frequency of double perforation. Examinations with the X-rays and sideroscope were indispensable to diagnosis. When the bits of metal weigh less than 3 mg the X-rays may fail, but Hertel's sideroscope points them out if they weigh as little as  $\frac{2}{10}$  mg, provided that they contain iron. Both methods fail when many splinters have entered simultaneously, and in cases of double perforation. Magnet extractions of fragments of steel are considerably less successful in war than in peace; the writer states that in peace he had eight per cent. of failures, while in war they have amounted to thirty per cent., and thinks this is due to the facts that on the average the wounded eyes are operated on later in war, that the force of impact was greater, and that double perforations were more frequent. Steel splinters from bombs are more irregular and jagged, and had more admixtures, especially with phosphorus, nitrogen, and manganese, which made their ability to follow the magnet less than that of splinters from steel tools, such as are met with in times of peace. Repeated observations seem to show that the admixtures favor a rapid onset of siderosis. Bits of copper and brass are successfully removed in a certain number of cases, yet half of the eyes subsequently are lost from infection, detachment, and other particles which have not been removed. Bits of stone were removed from the anterior chamber, but in only two were the eyes saved after removal from deeper portions. No attempt was made to remove the foreign bodies in other cases in which the eyes had been wounded by bits of stone, because they could not be located accurately. Powder driven into the eye was well borne in part of the cases. Bits of glass were almost always infected. Bits of straw and wood could be removed only from the anterior chamber.

K.

PINCUS (85, **Shot wounds of the occiput**) has studied twenty-two, mostly old, cases of visual disturbance after shot wounds

of the occiput. He found an optic neuritis in only three. The intensity and extent of the functional trouble are often disproportionate to the demonstrable traces of the external injury. The typical picture of homonymous hemianopsia was furnished by eight cases, but almost never so clearly as is commonly the case in diseases of the occiput. One case of wound of the left occipital lobe had optic aphasia and right-sided hemianopsia, which did fairly well after the shot had been removed. In five cases a hemianopsia inferior was present in varying degrees, only once in consequence of a typical cross shot. In one patient, who presented peculiar symptoms of fatigue, relatively darkened portions of the visual field became absolutely dark during an epileptoid fit. Once a hemianopsia superior could be demonstrated in the form of symmetrical scotomata in the left upper quadrant; in this case the X-rays showed a piece of a bomb in the optical radiation, but not in the cortex. When the visual centers in both sides of the brain were wounded, symptoms of disturbance of the higher visual faculties came more into the foreground; in two cases macular vision was preserved, while in two others it was lost although the greater part of one half of a visual field was saved. Symptoms of fatigue the author thinks are not always to be considered hysteroneurasthenic complications, but in many cases to be the expression of organic injury of the visual centers. Not only the perception, but also the correct projection of strong lights, is found to be preserved in the blind portions of the field. On the other hand, he knows of cases in which the defects of the visual field were of not so high a degree, but suffered from extraordinarily severe disturbances of the higher visual faculties.

K.

UHTHOFF (86, **Injuries of the central visual tracts and centers by shot wounds of the skull**) has observed forty cases of shot wounds of the occiput, three-fourths of which were complicated by hemianopic visual troubles. The cases were grouped into those having bilateral, monolateral, and no hemianopsia. The number of those with bilateral hemianopsia was greater than that of those with monolateral. Permanent total blindness was not observed after these occipital

wounds, except in one case in which a complicating meningitis induced a neuritic atrophy of the optic nerve and blindness. In only one case was the impairment of vision great enough to cause the patient to be classed with the economically blind. Four of the patients died. Hemianopsia inferior was much more common than hemianopsia superior, of which there was only one case. Only quite isolated examples of disturbances of the visual field which were not hemianopic in character were met with in this series of wounds of the occiput. Primary transient blindness, or great visual troubles which later improved, were very common, likewise primary loss of consciousness. The ophthalmoscopic condition was usually normal. Neuritic signs at the entrance of the optic nerve indicated a brain abscess in many cases, but in others this complication was absent. On the whole, little was seen of pupillary and muscular disturbances. Hemianopic hallucinations were observed in a few cases. With the exception of one, all of the patients were rendered unfit for further service in the field.

K.

After a brief explanation of the term psychogenous injuries as a collective name for hysteria, neurasthenia, and traumatic neurosis, and remarks concerning the existence of a sharply defined clinical picture known as traumatic neurosis caused by organic cerebral changes, OLOFF (84, **Psychogenous injuries of the eyes in war**) discusses the causes which induce these conditions in the present war. The principal part is played by the drum fire, explosion of bombs, and other severe explosions, that have not inflicted any physical trauma. In rare cases a psychogenous injury is grafted upon a wound, which cannot be explained through corresponding organic changes. The clinical picture is characterized, as a rule, by great diversity of symptoms, yet is not to be differentiated in its essential forms from the diseases met with in times of peace. In the foreground stand complicated motor signs of irritation and deficiency. Sudden deafness, complicated with dumbness, is not rare. Markedly few, on the contrary, are the psychogenous injuries of the eyes caused by battle thus far observed. The cases which have been described were all local phenomena

of a general hysteria with the usual hysterical symptoms outside of the eyes. Oloff reports two cases, in both of which the cause was great psychical high tension, due in one to the explosion of a bomb in the trench. In both of these cases the typical hysterical symptoms were confined to the eyes, and showed themselves pre-eminently in the form of spasms of the ocular muscles. Other hysterical symptoms outside of the eyes were completely wanting. It is also worthy of note that both of the patients were large, strong men, who had never before suffered from any nerve trouble. A hereditary taint could be determined in only one. These cases confirm the neurological observation already made in this war, that here and there healthy persons who have no hereditary taint may become hysterical in consequence of great mental exhaustion and shock, due wholly to the weight of the catastrophic occurrences of the war, and that hysteria may run a monosymptomatic course. OLOFF reports several other cases in which the psychogenous injuries of the eyes were local phenomena of a general hysteria and presented nothing special in their courses. The prognosis of psychogenous battle injuries of the eyes corresponds in general to that of war hysteria, and must be considered less favorable as regards the final result. He therefore recommends that such patients should not be sent again to the front, but to discharge them as unfit for service, as they are perfectly competent to perform their civilian duties. Some could be utilized for garrison duty.

K.





## ARCHIVES OF OPHTHALMOLOGY.

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### DISTORTIONS OF THE VISUAL FIELDS IN CASES OF BRAIN TUMOR

(*Fifth Paper*)

### CHIASMAL LESIONS, WITH ESPECIAL REFERENCE TO HOMONYMOUS HEMIANSOPSIA WITH HYPOPHYSEAL TUMOR.<sup>1</sup>

By CLIFFORD B. WALKER, M.D., AND HARVEY CUSHING,  
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(*With seventeen figures in the text and two illustrations on Text-Plate VIII.*)

IN the preceding paper of this series especial stress was laid on the bitemporal defect of vision associated with tumors in the interpeduncular space, particularly on those of hypophyseal origin. It was then stated that in a series of 148 patients, observed up to July 1, 1914, with outspoken manifestations of hypophyseal involvement—hyper- or hypopituitarism—101 had shown neighborhood symptoms of tumor, and of these 101, the visual pathway in 81 had become implicated.

These 81 cases, furthermore, were subdivided into three groups in accordance with the distortions of the fields at the time of the patients' admission to the hospital: (1) those exhibiting a typical bitemporal hemianopsia or a tendency in that direction (26 cases); (2) those exhibiting a typical homonymous hemianopsia or a tendency in that direction

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<sup>1</sup> From the Peter Bent Brigham Hospital, Boston, Mass.

(12 cases); (3) those in which complete blindness in one eye was present on admission, the other eye either being involved not at all or showing some constriction of nasal or temporal field (35 cases); and (4) those which presented bizarre fields of indefinite and irregular outline which were impossible to satisfactorily classify (8 cases).

In the succeeding three years to July 1, 1917, there have been 123 additional cases. The entire series at present represents 271 cases, of which 183 showed tumor manifestations and 148 showed field disturbance. This disturbance was bitemporal in 47 cases and homonymous in 22 cases. The remaining 79 of those showing field disturbances were blind in one eye, so that it could not be positively determined to which group they belonged.

From these statistics it is notable that bitemporal defects occur only about twice as often as homonymous defects in this group. That is, of the 148 cases showing field defects 14.8 per cent. are definitely homonymous in tendency, while 31.7 per cent. are definitely bitemporal.

It is with the twenty-two cases exhibiting definite homonymous defects, together with three cases in the amaurotic group, which were known from the history to have originated from a lateral rather than from a mesial implication of the chiasm, that this paper will deal.

Although attention has been called to the fact by others that with pituitary tumors of one sort or another homonymous hemianopsias may occur, it is nevertheless regarded as an unusual consequence of these lesions, and few ophthalmologists or neurologists venture upon a diagnosis of a lesion in this vicinity in the absence of a bitemporal defect, and indeed unless the bitemporal defect is fairly symmetrical.

In our preceding papers<sup>1</sup> especial emphasis was laid upon the character of the advance of the field defects toward the textbook type of bitemporal lesion, with vertical meridian bisecting the field, and it was shown that the advance is a fairly characteristic one, beginning, as a rule, in its earliest form

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<sup>1</sup> Cushing, H., and Walker, C. B.: "Distortions of the Visual Fields in Cases of Brain Tumor," *Brain*, 1915, xxxvii., 341.

Walker, C. B.: "Contribution to the Study of Bitemporal Hemianopsia," *ARCH. OPHTH.*, 1915, xliv., 369.

with an upper temporal loss of colors or very small visual angle test objects and progressing until merely an excentric patch of vision remains in the nasal field before blindness occurs.

Bitemporal hemianopsia was regarded therefore as a condition characterized by progressive changes in the temporal peripheries rather than by any particular fixed type of field defect. This seemed the more necessary for the reason not only that homonymous defects were almost equally characteristic of hypophyseal lesions, but for the reason that in many instances it was almost impossible to tell, given a certain stage and without the opportunity to observe fluctuating conditions by a series of charts, whether the condition actually was approaching or had gone through a bitemporal or an homonymous change.

Thus with an upper temporal defect in one eye and normal vision in the other, either type of bilateral field defect may follow: on the other hand, with an upper temporal defect in one eye and with the disturbance in the other gone on to blindness, either type of bilateral field defect may have preceded. Some of the better examples of these things occur in this series of homonymous distortions, and we were at first tempted to include them in the same report with the bitemporal cases.

On the other hand, a nasal defect in one eye gives much better presumptive evidence of an incipient homonymous defect though it may be difficult to determine whether it is of anterior or posterior origin with respect to the primary centers.

Here we would be greatly assisted in making a differential diagnosis if the Wilbrand prism test and the Wernicke pupillary reaction test could be used according to their theoretical conception. But as we have shown in previous communications,<sup>1</sup> the Wilbrand test is not so much dependent on the existence of a definite reflex arc as upon habit formations and psychological phenomena with respect to ocular movements

<sup>1</sup> Walker, "Topical Diagnostic Value of the Hemiopic Pupillary Reaction and the Wilbrand Hemianopic Prism Phenomenon," etc., *Journ. of the A. M. A.*, 1913, Pt. 2, vol. lx., p. 1152; "Observations on the Topical Diagnostic and Psychiatric Value of the Wilbrand Test with a New Clinical Instrument," *ARCH. OF OPHTH.*, 1915, vol. xlv., No. 2, p. 109.

with hemianopsia present. Accordingly we consider the Wilbrand test unreliable. The Wernicke test or hemiopic pupillary reaction also has failed to come up to expectations. We have found the hemiopic pupillary reaction present in all cases of anterior hemianopsia where the intrinsic pupillary mechanism was functioning well, but also it has been found present in hemianopsias of known posterior origin. The reaction may often be somewhat less marked in posterior than in anterior lesions, but in only one or two cases in a large series has the hemiopic pupillary reaction been absent with good intrinsic pupillomotor mechanism. Possibly it is true that most posterior lesions disturb the primary centers sufficiently to provoke the reaction so that it is only absent in isolated cases of the most uncomplicated type of posterior lesions.

Further light on the differential topical diagnosis may be supplied by certain characteristics of the field defects to an extent proportional to our knowledge of the mechanism of production of such defects. A few remarks on this subject before the presentation of cases may aid in the discussion.

#### MECHANISM OF CHIASMAL HEMIANOPSIA.

A very useful device to study the effects of various disturbances about the chiasm may be made by tearing away the strands of a piece of gauze until only a dozen or so fibers are left interwoven at the center. These may be taken to represent the crossed fibers and colored differently from other fibers laid along each side to represent the uncrossed fibers. The study of strains and tensions at various points in such devices, together with the study of autopsy specimens and X-ray, has led us to certain ideas about the action and reaction of forces in the chiasmal regions.

To take the simplest case first, we may consider bitemporal hemianopsia due to a symmetrical struma of the hypophysis. Here pressure is exerted on the lower part of the chiasm usually rather behind the mid-portion, so that the chiasm may be elevated and ride saddled into the upper anterior surface of the growth. An upper temporal defect begins, but does not cross the vertical meridian, although the nerve fibers representing each side of this meridian must be exposed to the same pressure

from the growth being practically equidistant from the same. Yet the crossed fiber suffers while the uncrossed fiber escapes. Hence pressure alone does not account for the result. Tension and strangulation of crossed fibers, because of their interwoven condition in their decussating region, are the additional factors. This is well seen in the model when a similar pressure is made. The crossed fibers are put under tension and strangle each other, while the uncrossed fibers are free to move away slightly, thereby receiving practically no tension or pressure, although lying possibly side by side with the crossed fibers. The tension of course augments the pressure and vice versa, and both increase the strangulation effect on the interwoven crossed fibers. Indeed it would seem that the strangulation action really affects the physiological block because we have seen the tracts themselves considerably stretched without being blocked correspondingly in function.

This process continues until complete bitemporal hemianopsia results, and then the progress of field failure may hesitate for some time. This hesitation simply represents the amount of additional pressure or distortion necessary before the uncrossed tracts may be in some way affected by the increasing tumor. It is evident from our specimens that several mechanisms and several types of field failure may then take place. The relations of the anterior clinoid processes and the tough dura stretched between them may act as points and lines of counter pressure against which the nerves are pressed or dragged. The remaining nasal field more often seems to shrink a little faster from below than from above, as might be expected to result from pressure upward against bone and dura of the foramen. There is no definite rule, however, and the nasal field may shrink up in a variable manner or quite symmetrically.

While these general considerations may account for much of the phenomena of bitemporal hemianopsia there remains to be explained the early occurrence of scotomata of the cæco-central type and the temporal island which we have previously described.<sup>1</sup> The former may be the result of hypersensitivity or possibly toxic œdematous action or both. The only

<sup>1</sup> Walker, C.B.: "Contribution to the Study of Bitemporal Hemianopsia," ARCH. OPHTH., 1915, xliv., p. 369.

explanation of the temporal island we have to offer is that it may result from the "knee"<sup>1</sup> of crossed fibers which loop far forward to the opposite side of the chiasm. Such a loop would act to relieve the tension and strangulation which occur in other crossed fibers of more direct course, thereby preserving vision slightly longer in the part of the field represented by the loop or knee fibers.

The growths producing the bitemporal defects are usually of strumous character and not so hard, so large, or so rapidly growing as the tumors producing homonymous hemianopsia. If, however, the tumor is of firmer and more irregular nature it may lean decidedly toward one tract or the other. Of course if this occurs after bitemporal hemianopsia has been produced, we have finally blindness in one eye and hemianopsia in the other, so that it may be difficult to tell in what class of hemianopsia the condition belongs excepting if we have records or history of the onset. Such tumors may oscillate to a considerable extent so that one part of the field improves as other parts are damaged. Indeed we have one case that started with an early bitemporal defect which finally became homonymous, the temporal defect in one eye recovering as the tumor broke through its confines laterally and destroyed the tract.

From such points as these it is evident that we may estimate whether the tract is being damaged by a growth substantially to the outer side of the tract or to the inner side. Thus if the tracts are affected in the former manner the homonymous hemianopsia is apt to begin and perhaps always be more advanced in the nasal field, while in the latter case the onset is in the temporal field, possibly both temporal fields, and perhaps only later and more slowly is the homonymous nasal field affected.

It is evident that the conditions so far discussed may be fairly well defined, but in addition we find numerous variations even to the most bizarre, resulting from tumor invasion of this region. Sometimes when the variation is not too marked we can estimate the probable mechanism, but then again we may find at autopsy a distortion of chiasm, tract, and nerves so

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<sup>1</sup> Wilbrand and Saenger, "Die Neurologie des Auges," vol. iv., p. 147.

marked that it is perhaps only surmise to speak of definite mechanisms. Several factors may be found superimposed. In general it may be said that a great variety of distortions are possible about the region of the optic foramina and clinoid processes. The nerves and chiasm may have the appearance of being strangulated, pulled, or pressed at numerous points, any one or various combinations of which might produce the resulting field defect. At the same time, in extensive tumors the tracts and primary centers may be greatly distorted, giving additional possibilities for field defects.

From a study of our specimens it would seem that pressure immediately lateral to the chiasm is not nearly so effective in producing homonymous hemianopsia as pressure on the tracts farther back against the peduncles. Indeed the sharply homonymous field defect seems always to indicate tract lesions in this group of tumors, while the more anteriorly the tumor presses, the more bizarre may be the departure from the typical picture as the chiasm and nerves are distorted.

However, the chiasm and anterior portion of the tract may suffer marked distortion without showing marked field defect. Such an instance may be now presented in accordance with our plan of presenting the cases in the order of magnitude of the field defect. All the cases in the homonymous group cannot be presented in this paper, but only such instances as seem to show the more typical characteristics of the group.

CASE I (Surgical No. 61). *Acromegaly with tumor.*  
*April 16, 1913.* Mr. G. B. S., aged 34. Onset of acromegalic overgrowth between 20 and 25 years of age. Sugar in urine first noted six months ago. Polyuria noted for two years. Headache in morning for years, increasing recently. Impotence began three years ago, complete one year ago. Drowsiness two years. Occasional nausea and vomiting last six months. Disposition very irritable recently. Petit mal attacks during last year. There has been no subjective disturbance of vision whatever.

*Examination on Admission:* Typical example of well-advanced acromegaly. Eyes perfectly normal in appearance and function throughout. Fundi normal; lamina cribrosa clear in good-sized optic cup. No venous engorgement or tortuosity. No disk oedema. Fields of vision perfectly normal to 5mm disks, form and colors. Sella turcica shows a tremendous enlargement by X-ray.

*April 29, 1913. Operation:* Transphenoidal sellar decompression with partial extirpation of struma accomplished without difficulty. The microscopic examination showed a different structure from other tumors of the series. The cells were more spindle-shaped and more compacted.

Patient improved subjectively after the operation and returned to work in very satisfactory condition, but finally was brought back to the hospital, *Mar. 13, 1914*, in a stuporous condition, and died in spite of the free use of cardiac and respiratory stimulants.

At autopsy a tumor was disclosed as shown in the photograph (Fig. 1). After filling the sella turcica the tumor had evidently found a plane of low resistance laterally, and had invaded the temporal lobe. Although the chiasm was pressed decidedly in the opposite direction, no field defect was found with 5mm disks at perimetric distance. While it is possible that examination with the test objects of small visual angles might have shown some slight field defect during the last year, it must be noted that a relatively high percentage of acromegalics showing a large sella by X-ray do not show field defects. Evidently in this case an automatic decompression took place laterally at a pressure just short of that necessary to produce a visual defect. Very possibly, however, the same pressure in another individual may have given a marked field defect. Indeed the study of specimens leads one to believe there is almost as much individual variation in tolerance to pressure on the optic nerves as there is in tolerance to the various toxic agents such as tobacco, alcohol, etc.

The following case shows the initial stage of an homonymous hemianopsia due to a primary pituitary struma distending the sella and extending laterally sufficiently to irritate the uncinate gyrus. The tardy appearance of the field defect is again notable.

CASE 2 (Surgical No. 28132). *Hypophyseal struma with hypopituitarism* (adiposo-genital dystrophy). Early homonymous upper quadrantal defects in Stage I.

*July '23, 1911.* T. M. E., aged 36. *Complaint:* Uncinate seizures.

*Examination on Admission:* A fairly typical case of hypopituitarism, with enlarged sella, uncinate fits, diffuse adiposity, hypotrichosis, high carbohydrate tolerance, high blood-pressure, etc.

*Eyes:* No exophthalmos, diplopia, amaurosis, nor nys-



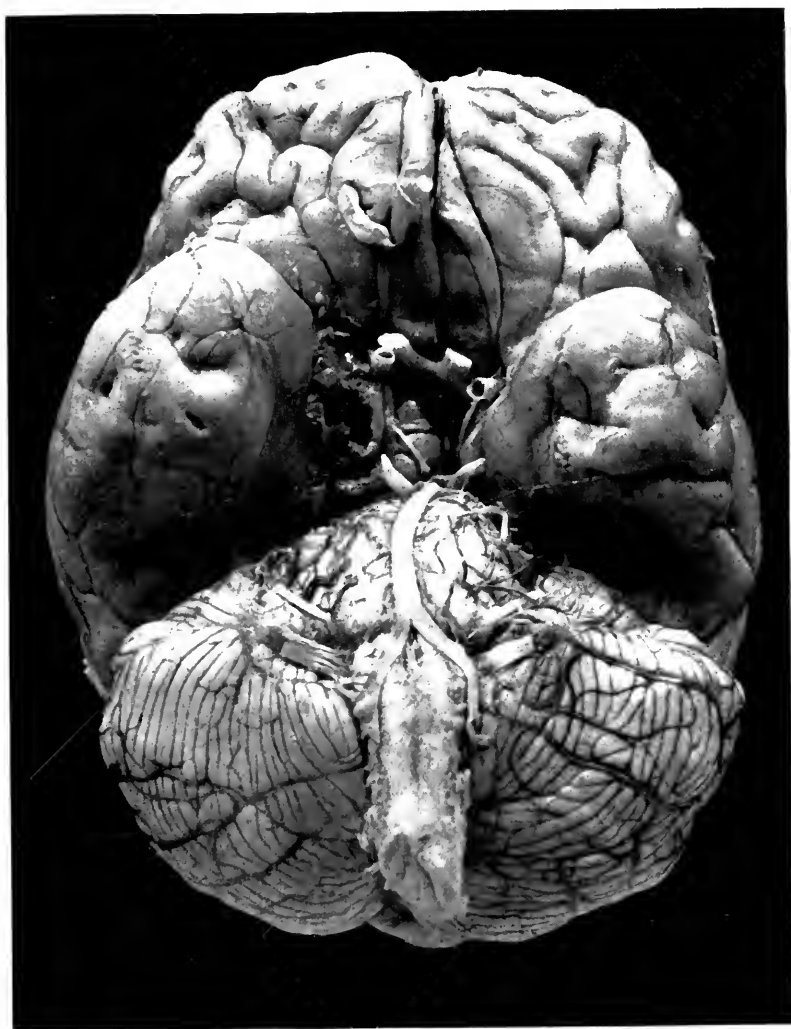


FIG. 1.—Case 1. Showing marked distortion of the chiasmal region by a sellar growth extending laterally without producing a homonymous field defect as might be expected.

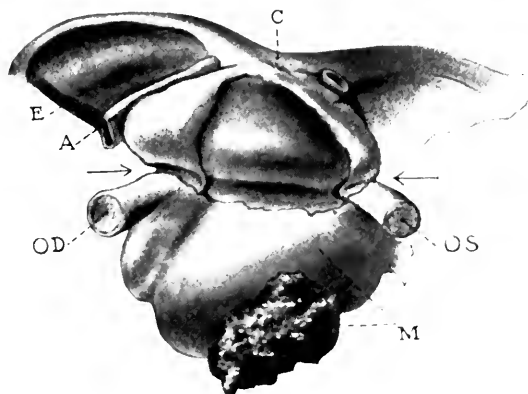


FIG. 13.—Case 5. Showing from in front the chiasmal distortion produced by an hypophyseal adenoma with intracranial extension. M is the intrasellar tumor mass, C the chiasm lying on top of tumor, OS and OD the optic nerves, which have been cut at the level of the arrow by the band of dura which connected the two anterior and widely separated clinoid processes. An artery (A) has also deeply cut the right nerve near the chiasm where it overlies on extension of the tumor (E) out into the right temporal lobe.



tagmus. *Fields* (Fig. 2) were supposed to be normal. V. O. D. 20/20; V. O. S. 20/20.

Glandular treatment, with improvement.

Nov. 6, 1911. *Readmission* for operation, owing to continued epileptiform seizures. Fields taken by Dr. Blair of Pittsburgh on Oct. 30th, and also those taken at the Johns Hopkins Hospital just before operation, showed a tendency toward a left upper homonymous color notch; but this was not appreciated at the time.

Nov. 9, 1911. *Operation*: Transphenoidal operation with partial extirpation of large struma.

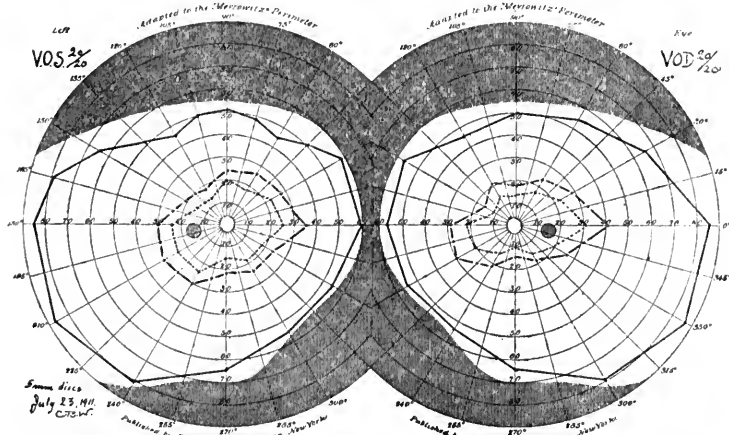


FIG. 2.—Case 2. Fields taken on admission and considered practically normal as far as a diagnostic lead was concerned.

A number of fields taken subsequently by ourselves and by Dr. Blair are practically the same as those before the operation.

Aug. 3, 1912. Patient returned for examination. *Fields* (Fig. 3) show a further advance in the left upper homonymous notch, making the homonymous character of the lesion unmistakable and certifying the insignificant changes of the earlier charts. An homonymous relative scotoma was also present. Fundi and acuity normal.

Sept. 23, 1912. During the past two months periodic lapses of memory have occurred in addition to an aggravation of the uncinate fits of gustatory character.

Oct. 5, 1912. Fields taken on return for examination show a slight decrease of visual acuity to V. O. D. 20/20 and V. O. S. 20/20—, and an increase in homonymous defects.

Oct. 8, 1912. Operation at Corey Hill Hospital. Second transphenoidal operation. Considerable bleeding at the time of operation but the patient made an uneventful recovery.

April 15, 1913. Further aggravation of memory disturbance and uncinate attacks. Disposition becoming more morose and apprehensive.

April 20, 1913. Eyes: Optic disks show a slight increase in pallor. Otherwise the fundus has not changed in appearance. Fields (Fig. 4) show slight increase in upper left

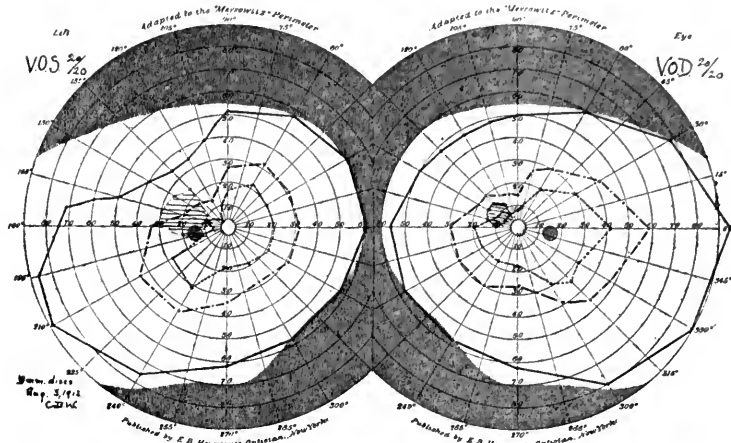


FIG. 3.—Case 2. Early left homonymous defect to color and form, with a relative left homonymous scotoma.

homonymous quadrantal defects: V. O. D. 20/20+; V. O. S. 20/20+.

April 26, 1913. Operation: Third transphenoidal operation. Difficult prolonged operation from which the patient did not rally sufficiently to have further field examination.

*Comment.* The foregoing observation is of unusual interest from many aspects. In the first place the patient, who for some ten years had consulted many physicians for headaches, adiposity, and impotence, began to have uncinate seizures, the character of which was recognized, but it was not appreciated that they were neighborhood symptoms of an hypophyseal tumor until the X-ray was taken which disclosed a greatly ballooned sella. It is remarkable, therefore, that a primary

hypophyseal struma reaching such a size as to irritate the uncinate gyri should have failed to implicate the optic nerves in the course of its enlargement.

Many fields were taken by ourselves and others, and all were regarded as practically normal, until the last fields here reproduced made a left upper homonymous defect unequivocal. Of course, looking back at previous fields (Fig. 2) in the light of later findings, it was possible to find traces of the condition, which, however, could not have been at the time safely inter-

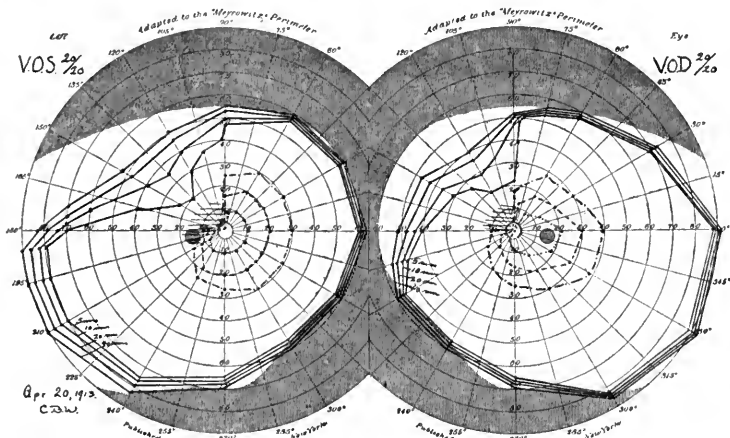


FIG. 4.—Case 2. Further increase in homonymous defect, showing response to large disks in defective region, indicating a state of physiological block and not atrophy.

preted. Here again examination with disks of very small visual angle would doubtless have brought out the real status of affairs much earlier. There are two other instances in the series of cases almost as early as this, but as we pass to the group having a stage of field defect more advanced but still short of complete homonymous hemianopsia, the number increases and the post-operative results have been more satisfactory, possibly because the visual disturbance occurred before the growth was too extensive.

**CASE 3** (Surgical No. 1140). *Acromegaly: polyglandular syndrome: diabetes.*

*April 11, 1914.* Mr. J. B., aged 47. Acromegalic

changes for past ten years, with feeling of "pressure in head." Headache extremely severe last six months. X-ray shows enlarged sella turcica. Glycosuria of 8 per cent., reduced after operation and treatment to 0.3 per cent.

*Eyes:* Slight exophthalmos. Palpebral fissures wide. Positive v. Graefe sign. Extra ocular movements normal. Convergence good. Joffroy negative. Pupils small, regular, and react to light and accommodation and consensually. *Fundi.* Disks have an injected appearance, but margins are sharp and lamina cribrosa is sharply stippled at the bottom of centrally situated optic cup.

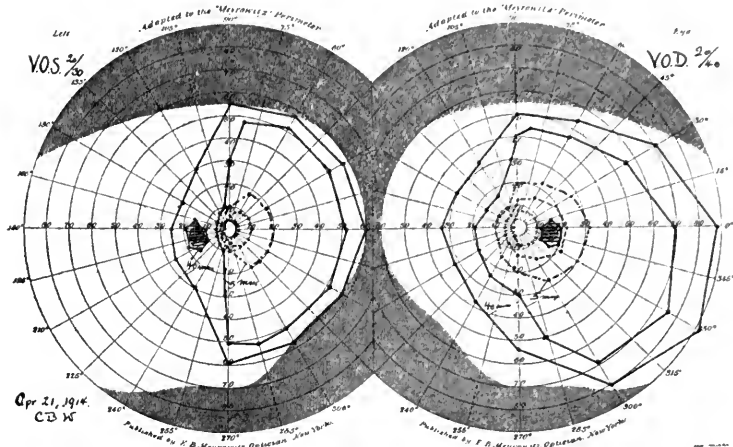


FIG. 5.—Case 3. Fields before operation, illustrating type of uniform shrinkage of homonymous fields.

*April 21, 1914.* Fields (Fig. 5) show an incomplete left homonymous hemianopsia. V. O. D. 20/40; V. O. S. 20/30. Headache and restlessness prevented prolonged examination.

*May 2, 1914. Operation:* Transphenoidal operation, second stage. Sellar decompression. Uneventful recovery.

*May 7, 1914.* Fields (Fig. 6) show a marked improvement in the homonymous defect, and a central vision above normal in each eye: V. O. D. 20/15; V. O. S. 20/15, which has been maintained up to the present time.

*Comment.* The action of the field defect in this case suggests that the lesion affected the right tract primarily and was possibly pressing from within outward in view of the relatively more advanced damage to crossed fibers representing the left temporal field. The post-operative recovery, very marked

and prompt as it was, substantiated this view, for the tumor must have been resting, in large part at least, on the floor of the sella in order to be released by a midline sellar decompression.

A variation from this type of rather uniform and symmetrical failure of the affected regions, is seen in the following case, where the gourd-shaped configuration so commonly seen in bitemporal cases is found in the homonymous type. Occasionally this conformation of defect is seen in the nasal

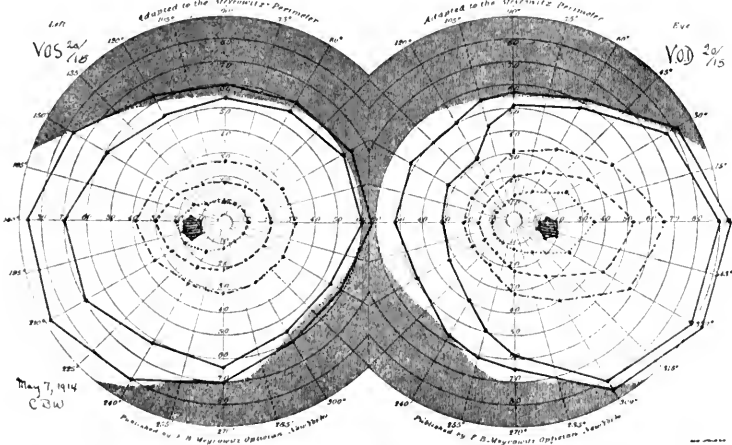


FIG. 6.—Case 3. Fields after operation, showing a type of recovery similar to the type of onset, in its uniformity.

field as well as in the temporal field of the homonymous field defect in this group, but it is more apt to appear in the temporal field only. It would seem to suggest a similar mechanism to that in the bitemporal group.

In the following case—one of typical outspoken acromegaly—the hyperplastic gland had undergone its so-called adenomatous malignant transformation and had presumably broken through its meningeal envelopes to the left of the chiasm.

For a time after the operation the condition in the right eye continued to advance, though there was an immediate improvement in the left. The final rapid restoration did not occur until X-ray therapy was instituted.

CASE 4 (Surgical No. 28552). *Acromegaly. Extrasellar extension of struma. X-ray therapy.*

Oct. 12, 1911. Miss F. J. D., aged 54. Headaches for eight years, boring and occipital in character. No nausea nor vomiting. Vision began to fail about twenty-one years ago, and she has never had the full use of her eyes since. Three years ago her eyesight grew decidedly worse; her appetite became enormous, and her hands and feet began to enlarge. She became somewhat mentally obtuse, and soon began to increase in weight. Failing lateral vision was first noted about one year ago; never diplopia nor

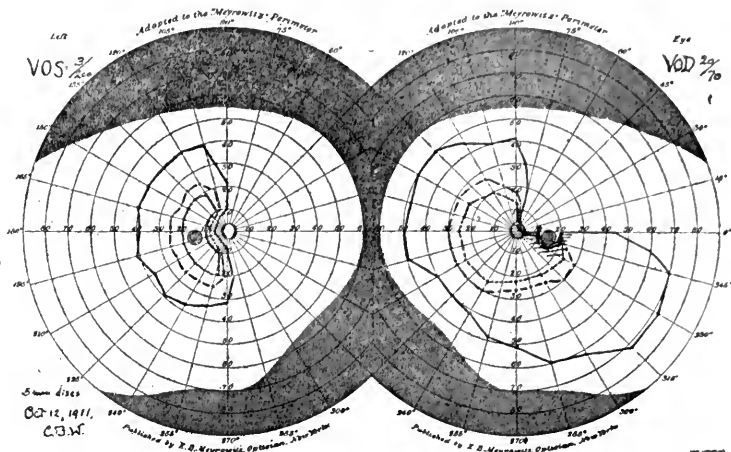


FIG. 7.—Case 4. Fields before operation, showing gourd-shaped tendency of temporal defect as in bitemporal hemianopsia.

exophthalmos. Polyuria for the past three or four years; also some epistaxis.

*Examination on Admission:* A typical case of acromegaly. X-ray shows an enlarged sella turcica.

*Eyes:* No diplopia, strabismus, nor exophthalmos. Pupils equal; react sluggishly to light but fairly well to accommodation. No nystagmus; movements complete. No v. Graefe. Consensual reaction very poor. *Fundi:* O. D., wide optic cup about 2 D. deep; normal color, perhaps slightly hazy nasally; vessels smaller than normal; lamina cribrosa clear. O. S., optic disk paler than normal, as in primary optic atrophy; lamina cribrosa sharp at bottom of wide optic cup 2 D. deep; fundus otherwise as in O. D. Fields shown in Fig. 7: V. O. D. 20/70; V. O. S. 3/200.

Oct. 13, 1911. *Operation:* Sellar decompression with



partial removal of fragments of hypophyseal struma. Good recovery, but during the course of a severe cold the visual

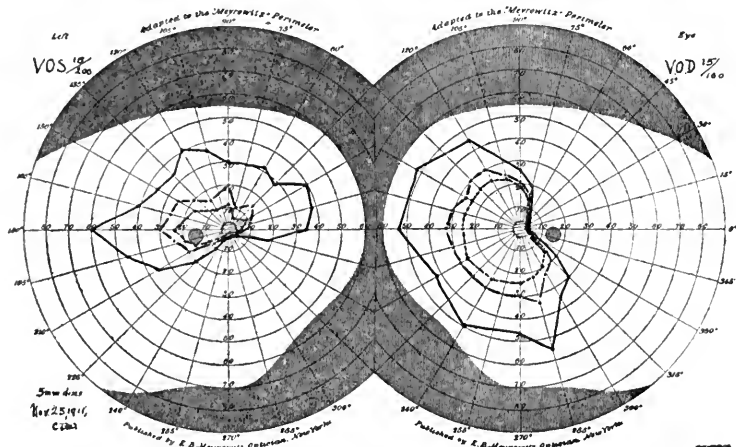


FIG. 8.—Case 4. Showing peculiar post-operative readjustment.

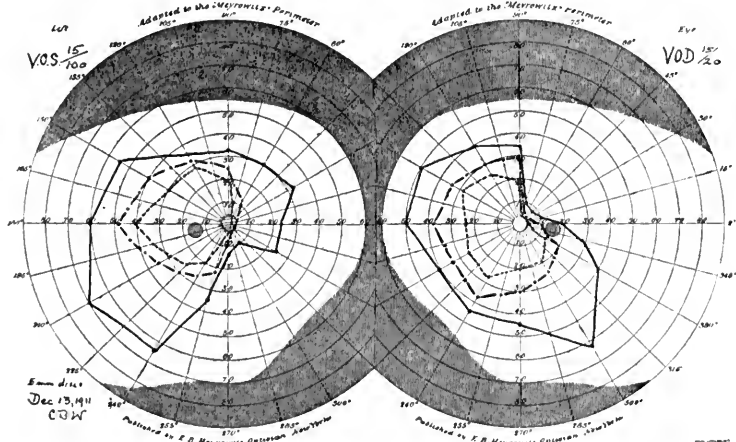


FIG. 9.—Case 4. Improvement in field extent, more marked in the eye most damaged before operation.

acuity sank to almost nil. Subjective improvement following this was fairly rapid, and on *November 23d* the fields shown in Fig. 8 were taken: V. O. D. 15/100; V. O. S. 15/200.

*December 13, 1911.* Fields shown in Fig. 9: V. O. D. 15/20; V. O. S. 15/100.

Dec. 30, 1911. Fields show further improvement, with one hazy island in temporal field of O. D. (Fig. 10): V. O. D. 20/20; V. O. S. 20/100.

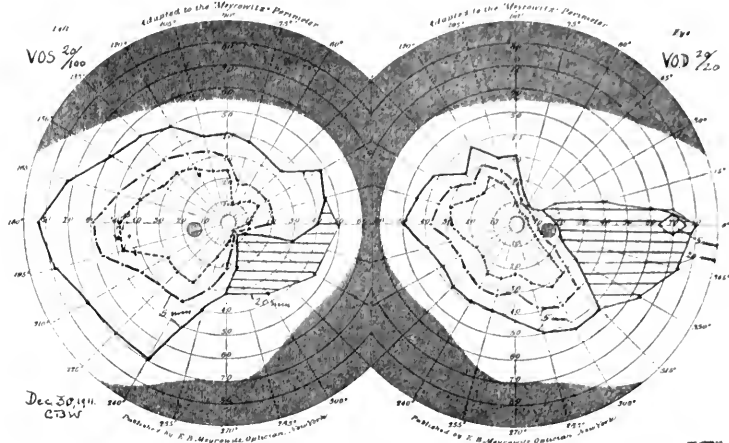


FIG. 10.—Case 4. Further improvement with marked tendency to persistent upper temporal and lower nasal defect. Note appearance of *temporal island* and manner in which larger disks were first used.

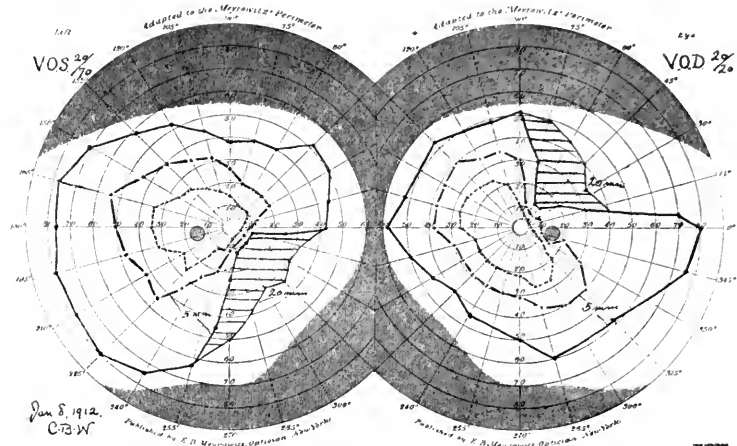


FIG. 11.—Case 4. Final record showing a very satisfactory visual improvement.

Jan. 8, 1912. Fields (Fig. 11) show still further improvement: V. O. D. 20/20; V. O. S. 20/70.

*Comment.* This patient described the early visual disturbance in a manner to suggest that the field defect was at first bitemporal in tendency but later became more homonymous in character. This is quite possible and indeed the shifting ability of the tumor and field defect in some of these cases is noteworthy in this series of fields. However, the more advanced nasal defect in the left eye would suggest that the tumor had broken through laterally to the chiasm so that pressure came to be predominately against the left tract, particularly the uncrossed bundle. To what extent this pressure was augmented by counter pressure at the foramen or clinoid process is uncertain except as interpreted by a study of the final readjustment in which we find a lower left nasal and an upper right temporal field defect. If this condition represents one of the stages during the onset, it could be accounted for by a pressure beneath the chiasm and anterior tract, and leaning laterally sufficiently to produce the temporal defect and pressing the left nerve against the sharp edge of the foramen above sufficiently to block the dorsal uncrossed bundle to give the lower left nasal defect. We have seen a tendency to this sort of diagonal field defect in other cases of this group, but never in the posterior homonymous group. Such a tendency with dyspituitarism speaks for a tumor extending far enough forward to involve the optic nerves either directly or with the aid of counter pressure in the region of the foramen.

Several of these points as well as previous statements concerning the degree of distortion necessary to produce certain field defects are well illustrated by the following case.

CASE 5 (Surgical No. 4310). *Acromegaly with tumor.*  
*Feb. 21, 1916.* Mr. J. H. D., aged 48. For past eight years gradual skeletal overgrowth with increase in weight, about ten pounds a year. Gradual increase of drowsiness and impotence over same period. Without headache, vision began to fail so that the right eye was practically useless four years ago, and the left eye became affected a year and a half later.

Two years ago the fields showed a complete loss of the nasal field of the right eye, with considerable shrinkage of the remaining temporal portion, while the left showed a large cæco-central scotoma and an upper temporal limitation

peripherally. Sella turcica enlarged. V. O. D. 20/100+; V. O. S. 20/30-. Operation was deferred at this time (two years ago), and since then the left eye has grown somewhat worse and the right eye slightly better.

*Eyes:* No imbalance, no exophthalmos. Right pupil slightly larger than left; both react sluggishly to light and accommodation. *Fundus O. D.* Disk margins sharp. Optic cup shallow. Lamina cribrosa can be made out but rather hazy. Decided pallor of disk, especially temporally, evidently atrophic. Fundus otherwise good condition. *Fundus O. S.* Similar in all respects except the disk, though pale, has much better color than in O. D. Fields (Fig. 12)

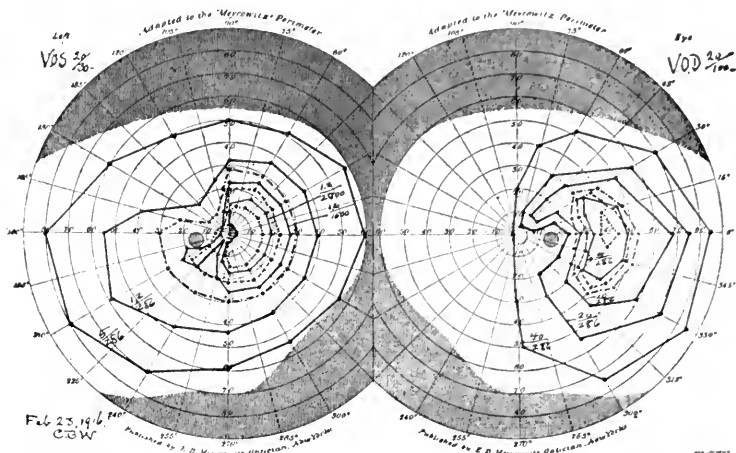


FIG. 12.—Case 5. Fields taken before operation with graduated disks, which give by the quantitative method much additional information.

show a left homonymous hemianopia incomplete in the left eye with decided central scotoma in the right eye: V. O. D. 20/100-; V. O. S. 20/30-.

*Mar. 3, 1916. Operation:* Transphenoidal. Partial removal of pituitary struma. Poor recovery from the operation. Much respiratory difficulty.

*Mar. 5, 1916. Respiratory failure. Death.*

From this case a specimen (Fig. 13) was obtained, showing a more marked degree of chiasmal and optic nerve distortion than was expected from the appearance of the fields. Probably the same amount of distortion in another individual would have produced a much greater amount of field damage. Such an individual variation might be due to variation in the firmness of anchorage of the optic

nerves at the foramen and consequent variation in ability to lengthen under tension, as well as to difference in ability to withstand pressure. Here again there is indication of greater damage below in the field of one eye and above in the field of the other eye.

These three cases will suffice to show the characteristics of the stages of homonymous hemianopsia between the onset and the stage of complete hemianopsia. It is notable that there may be a rather uniform general contraction of the homolateral fields or there may be a very symmetrical gourd-shaped stage produced from above or below. One eye usually is more advanced than the other, as in bitemporal cases. As the rind line is reached, central scotomata become bothersome, especially in the more anterior lesions. When a lesion is effective farther back in the tract, as in the following case, the field defect may be more sharply cut in the rind line and central scotomata somewhat more delayed though relatively present.

CASE 6 (Surgical No. 29395). *Hypophysis tumor.*

*Mar. 7, 1912.* Miss A. G., aged 27. Severe headaches, left temporal and frontal, always associated with nausea and vomiting for past two and one-half years. Six months ago, vision began to fail, until now cannot read. For past three months drowsy and stupid.

*Examination on Admission:* Fairly typical case of hypopituitarism. X-ray shows obliterated sella.

*Eyes:* Left pupil twice as large as right. Both react to light, accommodation, and consensually, but left is much more sluggish. Hemiopic pupillary reaction present. Never diplopia. Both eyes prominent, exophthalmos left. No nystagmus. Motion of left eyeball considerably limited.

*Fundus O. D.* Disk slightly elevated, 0.5 D. nasally. Optic cup not filled but lamina cribrosa cannot be made out. Vascular condition not greatly changed. Fundus otherwise normal.

*Fundus O. S.* Optic disk pale with sharp margins with optic cup present but indistinct lamina cribrosa, which has a glistening pallor, temporally suggestive of primary optic atrophy. Vascular condition and fundus otherwise as in O. D.

*Fields* (Fig. 14) show a vertically cleft right homonymous hemianopsia.

*Mar. 18, 1912. Operation:* Right subtemporal decompression. Lumbar puncture.

*April 1, 1912.* Fields practically unchanged. Fundi show no measurable swelling; otherwise as before.

*April 2, 1912. Operation:* Sellar decompression.

*April 16, 1912.* Fields show no notable change. Fundi show no notable change.

Choked disk is of comparative rarity in the hypophyseal group, due to the fact that the sheath of Schwalbe is blocked off early in the process, so that the usual hydrostatic phenomenon cannot take place, although cerebral pressure, with

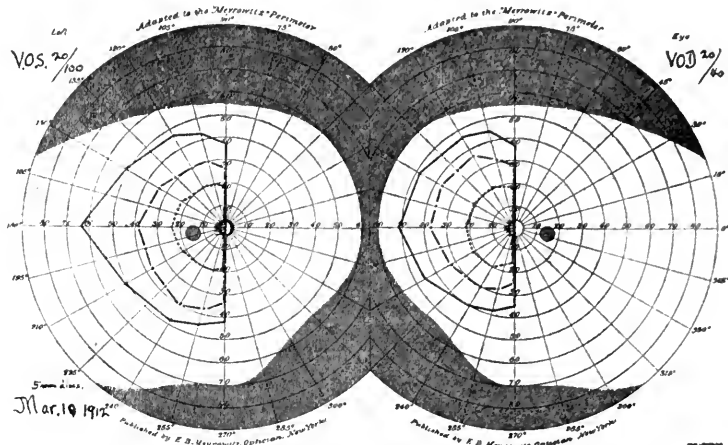


FIG. 14.—Case 6. Fields showing vertically cleft stage and relative central scotomata depressing the central vision,—a condition more common in anterior than in posterior homonymous hemianopsias.

severe headache, nausea, and vomiting, may later take place. When choked disk does occur it is usually moderate and speaks for a growth of considerable size well back in its major extension. However, we have seen a meningitis give the picture of a choked disk of low grade even in a case of bi-temporal hemianopsia, so that a hydrostatic pressure in the usual sense may not always be the exciting cause.

In addition to the preceding cases given as examples of the more or less characteristic progress of the defect in the homonymous group, we may give instances where one eye has become blind or nearly so before the other eye shows much or any damage. It may be difficult to classify such cases as bi-

temporal or homonymous in tendency. The deciding factors may be the character of onset in the blind eye obtained from the history or the previous charts, even though poor,—the position in the field of any trace of light perception in case the eye is not absolutely blind, and the character of any minor changes to be found in the good eye. In the following case all of these were available.

**CASE 7** (Surgical No. 3781). *Hypophyseal struma with dyspituitarism.*

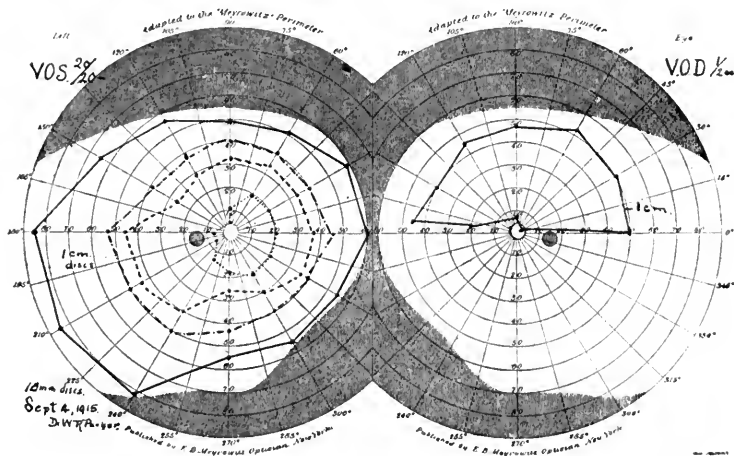


FIG. 15.—Case 7. Fields taken before admission,—equivocal, but with slight suggestion of homonymous tendency.

*Oct. 29, 1915.* Mr. I. H., aged 33, referred by Dr. Walter R. Parker of Detroit, Mich. An obese individual with characteristic manifestations of dyspituitarism. Frontal and temporal headaches, increasing in severity since onset one year ago. Three months ago discovered that vision in right eye was failing. Fields (Fig. 15) taken by Dr. Parker, Sept. 4, 1915, show a marked defect from below in the right eye, with a beginning upper temporal defect in the left eye. V. O. D. 1/200; V. O. S. 20/20—. The sella turcica was found to be enlarged by X-ray.

*Eyes:* No exophthalmos, nystagmus, nor muscular imbalance. *Pupils:* Right pupil moderately dilated and shows a very sluggish direct reaction to light. Indirect reaction good. Left pupil gives a good direct reaction, but a poor indirect reaction to light. *Fundi:* Both disks

are pale, but pallor of right disk is decidedly more marked so that a certain amount of atrophy may already have taken place in right optic nerve. The disk margins are sharp and pigmented. The lamina cribrosa is slightly blurred but is nevertheless readily seen. *Fields* (Fig. 16) show an upper temporal defect in the left eye with a central vision of 20/30, while in the right eye there is left only a small uncertain area of light perception in the upper temporal quadrant.

*Nov. 6, 1915. Operation:* Transphenoidal operation for pituitary struma.

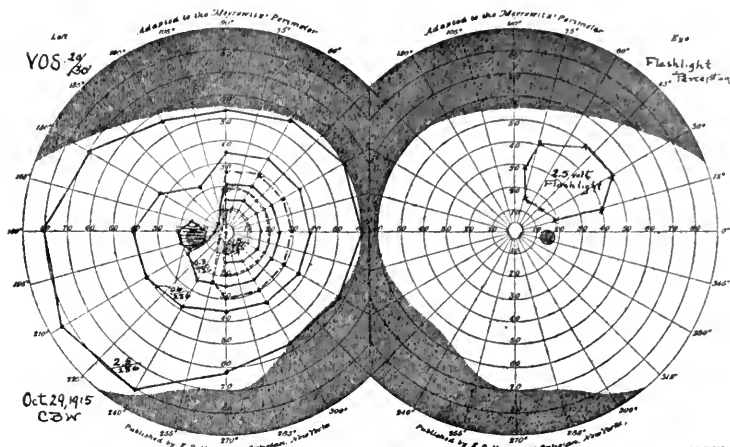


FIG. 16.—Case 7. Field taken before operation, showing definite homonymous tendency, as indicated by residual light perception in upper right temporal field.

*Nov. 10, 1915.* The return of vision in the right eye was extremely rapid, being easily the record not only for rapidity but also for the quality. The vision had returned to 20/20 in three days after the operation, and to-day, the fourth after the operation, the fields (Fig. 17) show an almost normal outline in the left eye, even to the finest disks, and in the right eye, except for a lower nasal defect, the visual peripheries are practically normal. Remarkably good central vision. V. O. D. 20/20+; V. O. S. 20/15-.

*Nov. 15, 1915.* Patient discharged in excellent condition.

*Jan. 12, 1916.* Report of Dr. Parker states that patient is back at work in no way inconvenienced, either by the lesion or by the result of the operation. A very slight lower nasal defect persists in the right field. V. O. D. 20/15+; V. O. S. 20/15+.



*June 5, 1917.* A communication from Dr. Parker shows that vision has held the same and the fields are normal to the 5mm disks.

*Comment.* The recovery of vision after operation in this case from practically blindness of the right eye to practically normal vision in four days is the most startlingly rapid recovery we have seen, though some others in the bitemporal group have approached quite closely to this record.

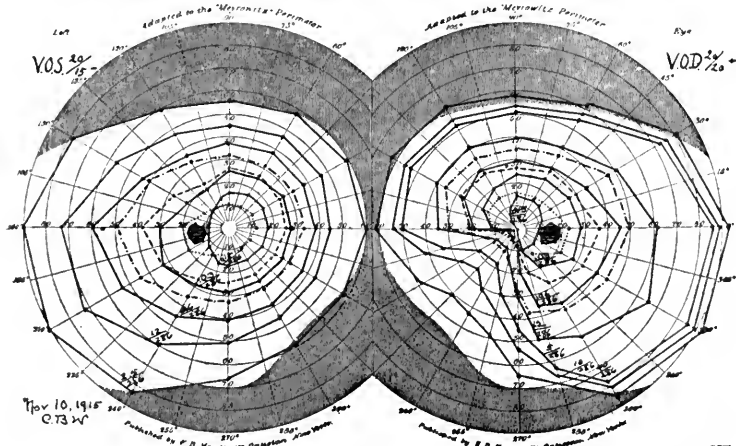


FIG. 17.—Case 7. Astonishing amount of field and central vision recovery in less than four days after the operation, establishing in this respect a record visual improvement for all transphenoidal operations done up to the present. Further improvement has been reported by Dr. Parker since patient left the hospital.

The question as to whether to classify this case on admission as bitemporal or homonymous could perhaps not be answered from the field taken previously (Fig. 15), though the nasal defect of the right eye seemed slightly more advanced than the temporal, suggesting a homonymous defect in tendency. The field taken on admission (Fig. 16), however, decided the question, since it was found that the small amount of light perception remaining in the right eye was located in the upper temporal portion of the field, and the manner in which the field later recovered left no room for doubt.

It is interesting to conjecture about the mechanism of the field defect in this case. The tumor without doubt, after

filling all the available sellar space and producing sellar enlargement by pressure, broke through to the right so that the right optic nerve was forced upward against the rather sharp edge of the foramen or the rough dural interclinoid margin. This concentrated counter pressure being more effective than the rather diffuse pressure from the tumor resulted in the more marked field defect from below. The transphenoidal decompression allowed the tumor to settle downward and the vision was quickly regained because of the release from acute strangulating pressure on the right optic nerve. Moreover, the duration of this acute pressure was short,—three months, which is a condition always favorable to a rapid recovery since atrophy does not become marked until six months or more.

Here again we see the tendency to an upper temporal and a lower nasal homonymous defect as in the previous case. Whether or not there is some rotation of the nerve as the chiasm is tilted upward on one side, to aid in producing such a result, is difficult to state though it would seem quite possible.

In addition to the foregoing rather typical instances of field defects in the homonymous group, we have those cases showing central scotomata sometimes of large size but without marked peripheral defect. When such a scotoma is present in one eye only and a temporal hemianopsia present in the other, as in the following case, we have a condition that may readily develop into a bitemporal or a homonymous hemianopsia.

CASE 8 (Surgical No. 4409). *Pituitary group. Dyspituitarism with struma.*

Mar. 12, 1916. A. O., aged 34. Severe frontal headaches every month or so for years, growing markedly worse in last three years and tending toward left side of head. Some nausea with these headaches in last six months. Loss of left lateral vision first noted  $2\frac{1}{2}$  years ago. Also loss of libido. Moderate polydipsia and polyuria. Inconstant diplopia for last year.

*Eyes.* Left pupil about  $1\frac{1}{2}$  mm larger than right; both regular in outline but left rigid to light and accommodation, direct and indirect, while right is active to same. Partial paralysis of left internal rectus giving slight external strabismus. Slight ptosis of left lid. No exophthalmos or nystagmus. *Fundi* normal except for slight increase of

disk pallor. Pallor of both disks of same grade. Lamina cribrosa sharp. Optic cup well formed.

*Fields* (Fig. 18) show a temporal hemianopsia in the right eye, and a large central scotoma in the left eye. V. O. D. 20/30; V. O. S.  $\frac{1}{2}$ /200.

*Mar. 20, 1916. Operation.* Transphenoidal procedure with partial removal of soft struma. Recovery from operation, followed by disappearance of all subjective discomfort.

*April 10, 1916.* Fields (Fig. 19) show considerable improvement: V. O. D. 20/20; V. O. S. 10/200.

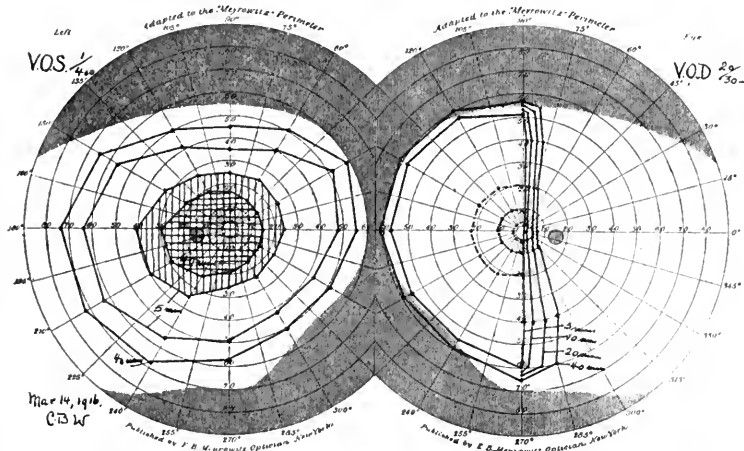


FIG. 18.—Case 8. Fields taken before operation, showing central scotoma in left eye and hemianopsia in right eye associated in such a way that further visual failure in the right eye may throw the case into either the homonymous or bitemporal group.

*Comment.* While the more advanced defect in the right temporal field suggests a mesially disposed pressure which would result finally in a bitemporal hemianopsia, there is also evidence in the tendency of the scotoma to remain slightly more extensive in the lower nasal field of the left eye after operation that a homonymous tendency is present. The presence of such scotomata as this speaks more often for a tumor than a struma, in which case lack of symmetry might be expected, resulting in a homonymous type of field. It is difficult, if not impossible, to explain a scotoma of this character on a mechanical basis alone, so that we find ourselves

reduced to the rather unsatisfactory explanations based on hypersensitiveness of the papillo-macular bundles or possible toxic actions.

#### GENERAL COMMENT.

Certain cases in this group have shown remarkable improvement in vision after the transphenoidal operation. Most striking of these is Case 7 where a blind eye recovered normal

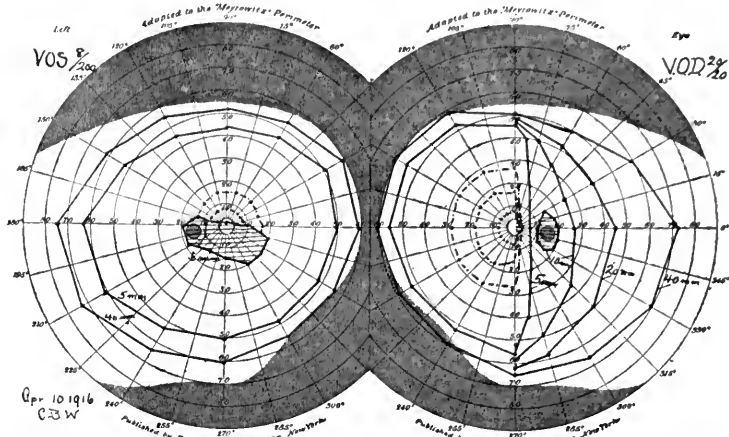


FIG. 19.—Case 8. Fields after operation, showing visual improvement. Slight evidence of homonymous tendency.

vision in three or four days with an almost complete field recovery. Case 4 also shows a slower but very satisfactory recovery of vision. But altogether, from an operative standpoint, the results are not so satisfactory in this group as in the bitemporal group as regards improvement in vision. This of course is accounted for by the fact that most of the lesions in the homonymous group are tumors rather than strumas and have a tendency to grow more wilfully and unsymmetrically.

It is notable that the extent and size of the tumors may not give a very exact idea of the amount of chiasmal distortion present. Thus in Case 1 a marked distortion of the chiasm failed to give any decided field defect such as might be expected. Also in Case 5, considering the amount of distortion and compression shown in Fig. 13, a considerably greater

field defect would not have been surprising. Possibly individual variation, not only in the response of the optic fibers themselves to insult but also in the firmness with which the optic nerves are held in their foramina, may have a decided influence on the resulting field defect.

While the operative results in the homonymous group of pituitary growths are not so uniformly successful as in the bitemporal group, because of the difference in the nature of the growths, nevertheless surgical interference has been amply justified by brilliant results in certain cases and in checking the failure of vision in others.

## HEADACHE AS THE RESULT OF EYESTRAIN.

By JOHN DUNN, M.D., RICHMOND, VA.

**Y**EARS ago the problem headache was led to the door of Ophthalmology and left there. Certain relations between the use of the eyes and the production of headache were noted, and upon these observations the art of applied refraction, including the determination and suggestions for treatment of muscular imbalance, was built up. So far as I know, however, ophthalmology has made no effort to answer the question: How does the correction of refractive errors relieve headache? Or, a step further back, the other question: What is headache?

To say that the use of glasses lessens the strain on the ciliary muscles and so relieves or prevents headache—even though true—is no answer at all. How does the use of the ciliary muscle produce headache any more than use of the deltoid or the peronei? If use, or over-use of the ciliary muscle produces headache, why does it not always do so? In the scheme of the human economy, nature plays no favorites. Mrs. Brown is forty and is both farsighted and slightly astigmatic; has used her eyes for close work as much as she chose to; has never worn glasses, and has never had a headache in her life. Mrs. Smith, aged forty, has an equal degree of farsightedness and astigmatism; has no imbalance of the external ocular muscles, and yet for years the slightest prolonged use of her eyes for close work, unless she was wearing proper glasses, has in a large proportion of the times resulted in headache. Mrs. Jones, aged forty, with the same external muscular conditions, and the same refractive errors as Mrs. Smith and Mrs. Brown, can use her eyes neither with nor without glasses that the result is not invariably headache. Baldly stated there is the problem.

To say that Mrs. Smith has a general condition which lessens the tone of the ciliary muscle, and that Mrs. Jones has some physical trouble which still further increases the difficulty of response on the part of the ciliary muscle to retinal-born impressions and demands, is to bring us a doubtful step nearer, but such an answer still begs the question: Why should use of the ciliary muscle produce headache and not use of the sartorius do the same? What relation has use of the ciliary muscle to the production of headache? Why not jaw-ache, or toothache, or earache? Why headache? The ciliary muscle is a structure outside of the skull. Headache is a disturbance which resides inside the skull.

Again, take the case of Mrs. L., aged 36, who has oblique hyperopic astigmatism of  $1\frac{1}{2}$  dioptries in each eye. To go without correcting glasses for even a short while results in headache, vertigo, and nausea. Wearing her glasses she is able fairly comfortably to do a full day's general work. Exercise of the muscles of her arms over the wash-tub produces with her neither headache, vertigo, nor nausea. Exercise of her accommodation, with the refractive errors uncorrected, produces all three. It is plain that the headache, vertigo, and nausea are not simply the result of a continual call on the brain cells for the origination and continued co-ordination of muscular impulses—in other words, of sustained attention. If so, the work over the wash-tub would cause them. The vertigo and nausea are capable of explanation as the result of excessive irregular, perhaps spasmodic, contractions of the ciliary muscle. The impulses thus originated reach the medulla and overflow into the vagus center (nausea and vomiting) and into the vestibular centers (vertigo). That this is probably the explanation of ocular vertigo and nausea, can be inferred from the ease with which vertigo and nausea can be aroused by disturbance of the fluid in one or more of the semicircular canals. Here the nerve impulses from the canals after reaching the triangular nucleus in the medulla over-stimulate the adjacent vagus centers and may produce pallor of the skin, sweating, nausea and even vomiting—but *not headache*. What is it that causes the headache? Again, take another of the symptoms found so frequently accompanying headache secondary to eyestrain. I have reference to what the patient

terms "nervousness." "Doctor, when I have one of my headaches, I get so nervous I could jump out of my skin." A temporary and transient symptom this is with some cases. With others, especially where there is a rather high degree of compound hypermetropic astigmatism with external muscular imbalance, the "nervousness" becomes, as the years go by, an almost constant symptom and the predominant one in the patient's mind and in that of the minds of those with whom he or she is surrounded. To what is this nervousness due? As I see it, there is only one satisfactory explanation, and that is that it represents a reflex instability of the physiological intracranial pressure, the predominating phases of which is a pressure above normal. The extreme phases of this nervousness are to be found in certain cases of acute intracranial inflammation and injuries.

What is headache? A pain in the head? That will not do. We are after a definition which explains, not one that defines. We are chasing game in the fields of medicine, not in the pages of a dictionary. Why should the head ache after use of the eyes? Because the eye is innervated by nerves which have emerged from the brain? So are muscles of the auditory drum membrane and yet response of the tensor tympani to sound waves does not produce headache. What brings about the headache? What are the actual intracranial conditions which cause the headache? How do these conditions come into existence?—in some cases so easily, in others with such relative difficulty.

Such is the problem before us and I have presented it in the foregoing form that I might, as far as I could, divest it of all its superfluities and so prepare the question for the best answer I can give.

*Headaches, inclusive of those due to injuries and to tumors, are a manifestation of increased intracranial tension.*

Let us examine the headaches in a case of brain abscess and in one of brain trauma.

Jno. K., aged 26, showed symptoms which suggested the forming of a right-sided cerebellar abscess. Three or four days later he became wildly delirious, his pulse dropped to about 30 to the minute, and he began to scream repeatedly at the top of his voice as if in the most intense agony. The



bone was removed over the right half of the cerebellum. A deep-seated abscess was found and evacuated. Immediately following the opening of the abscess, the pulse jumped to 140 to the minute. Within a very short while the pulse was regular and 84 to the minute; the patient was perfectly conscious and the pain in his head had disappeared. The severe headache was not due to the presence of the infectious material within the cerebellar substance, for although the abscess was opened and much thin purulent matter ran out, the cerebellar substance immediately closed in upon the abscess cavity, obliterating it and the tract by which the pus escaped. The infection remained, as was proven later by a reaccumulation of pus, pressure upon the respiratory center, and death—but *no further headache*. The headache was not one due to meningeal inflammation, because of its disappearance practically immediately after the opening of the cranial cavity. It may be safely said that the headache was a manifestation of an acute rise of intracranial tension, and in this case one due to blockage within the region of the lower water-bed, probably of the iter itself.

Again take the case of Mr. L., who fell from the roof of a shed, striking the right temporal region against a pile of bricks. The next day he was wildly delirious, screaming with pain and unconscious. The removal of the right temporal plate and ligation of a branch of the middle meningeal artery and evacuation of a clot resulted in the restoration of consciousness and disappearance of both delirium and headache in a few hours. It was not the injury to the skull cap which induced the symptoms nor the injury to the dura, nor the rupture of the middle meningeal artery. It was the increasing intracranial tension, for none of the symptoms followed immediately upon the receipt of the injury.

Similar instances of headache following upon acute rise of intracranial tension could be multiplied indefinitely. The above two have been mentioned here to show that the factor in the production of the headache in such cases is not the cerebritis; nor injury to the dura or to the bony cranium, or to the meninges, but to a rise in the intracranial tension, a subject about which a great deal has been written. The medical literature of this especial subject has to deal with that type of intracranial tension which accompanies brain tumors, abscess, meningitis, and cranial injuries. Relatively little has been said about what may be called *physiological increased*

*intracranial tension, its cause, and method of production and regulation.*

What constitutes a normal intracranial pressure? The intracranial pressure is normal so long as it is equally distributed over all parts of the brain surface and admits of an intracranial circulation of the blood just sufficient for the full needs of brain metabolism.

What is cerebrospinal fluid? It is a "colorless, limpid fluid of low specific gravity, among whose grosser chemical characters are its low protein content and the presence of a definite reducing body known to be glucose." Wegefardh and Weed (*Journal Med. Research*, vol. xxxi., 1914-15). It is elaborated chiefly by the choroid plexuses in the lateral ventricles, and is found intracranially in the ventricles, in the water-bed of the posterior fossa, and generally in the meshes of the pia-arachnoid.

How does it escape from the interior of the cranium? Key and Retzius (1876) claimed this occurred through the Pacchionian bodies into the longitudinal sinus.

Dandy and Blackfan (*Am. Journal of Diseases of Children*, vol. viii., 1914) as the result of their experiments came to the conclusion that it found its way out by "the exposed capillaries of the pia-arachnoid" and so into the veins.

Weed (*Journal of American Research*, vol. xxxi., 1914-15) says "the anatomists (J. Mestrezat, W) are agreed that the arachnoidea is non-vascular. Weed further says he has never observed any evidence of a capillary bed in the pia. Weed came to the conclusion that the absorption was through the arachnoid villi wherever found. The distribution of these villi in order of frequency is—superior longitudinal sinus, transverse sinus, cavernous sinus, and *venæ meningiæ mediæ*. That these villi communicate with the fixed venous channels does away with the variations in pressure which occur in the free veins, a precaution which only serves to accentuate the extreme delicacy of all the brain processes. This anatomical provision lends weight to the correctness of Weed's observation. Some of the cerebrospinal fluid escapes from the spinal canal. Weed's experiments seem to show that it is relatively slight in amount. By what passages this escape is made is unknown. The problem of the secretion of the cerebrospinal

fluid, however, is not solved by having discovered its source and its channels of escape. Nor do the results of analyses answer all the questions.

Weed (vol. *cit.*) says: "The mechanism of the cerebral pressure relations is such that the cerebrospinal fluid pressure is continually being reduced to that of the venous circulation and, in consequence, there must be a constant absorption of the fluid to compensate for the constant secretion." What is it that regulates both absorption and secretion? The cerebrospinal fluid makes the exact intracranial pressure adjustment possible, but there must be something which determines the volume of output from the choroid plexuses and the correspondingly controlled passage into the fixed veins. What is it that regulates the degree of permeability of the fluid itself? These things are not merely mechanical. They must be presided over by an extremely delicate nervous mechanism. The slightest undue pressure upon the cortical cells calls for instant adjustment. The position of the hypophysis and the fact that its posterior lobe secretion passes into the third ventricle suggests that its rôle is in part to regulate the intracranial pressure. Bell says that even though "the secretion from the pars nervosa does pass into the cerebrospinal fluid, as has been asserted, there is not the slightest evidence that this is essential, beneficial, or even the normal method by which the internal secretion is taken up by the animal economy." (*J. A. M. A.*, p. 1698, 1917.) This may and probably does apply to the activities of the anterior lobe. Indeed, we have reason to infer this from consideration of the other glands of internal secretion. The output of no other of these glands is poured into a space similar to that into which the posterior lobe empties its secretion. Why has the posterior lobe its peculiar channel? The cranium, the eyeball, and internal ear have each a water system, the prime object of which would seem to be to make possible an even pressure upon the cortical cells of the first and upon optic and auditory and vestibular terminals of the other two. If we look at the mechanical arrangement of the water system of internal ear, we see that it is so constructed that under normal conditions the pressure upon the auditory and vestibular terminals therein varies with the intracranial pressure. At first glance it

would seem that no such provision has been made for the eyeball. Wegefarth's (*J. of M. Research*, vol. xxxi.) experiments, however, would seem to prove that "by varying the pressure relations existing in the cranium and in the eye" there is "a direct connection of the two fluid spaces, at least for true solutions." It would seem further, however, that under normal conditions the water system of the eye is sufficient to regulate the pressure upon the optic terminals without the assistance of the intracranial system *save for one thing*, and that is, it cannot furnish to the aqueous that something whose presence we may infer is necessary to regulate the permeability of the aqueous after its secretion. There must be some provision for controlling both the output and the escape of the cerebral fluid. The distal situation of the arachnoid villi and the driving force of the intracranial circulatory impulses are some of the mechanical factors; but we are dealing with a problem different from a pumping station and hydraulic outlets. The intracranial pressure has to be rapidly regulated within almost infinitesimal limits to meet the normal requirements of the cortical cells. There must be some reflex arrangement whereby the permeability of the cerebrospinal fluid is made to vary. It is difficult to imagine a way by which this can be done save by the addition to the output of the choroid plexuses of a substance to accomplish this end. It has been proven that the arachnoid villi are permeable to aqueous solutions, just as they are permeable to the cerebral fluid itself. This merely shows that the arachnoid villi would mechanically admit the passage through them of a cerebrospinal fluid deprived of its regulative agent and that the purely mechanical part of the intracranial water-way is sufficient to prevent a very high intracranial tension. It by no means proves that a purely watery cerebral-spinal fluid is sufficient for the normal processes of the cerebral tissue. Indeed, there are reasons for believing it is altogether insufficient. The position of the hypophysis and the fact that its posterior lobe secretion finds its way into the third ventricle and is mixed with the output of the choroid plexuses is highly suggestive that this secretion is the substance which has an influence upon the permeability of the cerebrospinal fluid. Deprive this fluid of the posterior lobe secretion and the brain processes go on without its regula-

tive action, but there appear in the course of time many disturbances of bodily growth; these latter, I believe, are to be attributed in part to an imperfectly regulated intracranial pressure. That the choroid plexuses will continue to secrete in the presence of a non-functionating posterior lobe seems to have been proved. It has equally been proved that the bodily processes do not progress normally under the same conditions. It seems more than probable that the activity of the posterior lobe responds reflexly to the general intracranial pressure, *i. e.* of the pressure upon the cortical cells. *It is probable also, that continued higher than normal intracranial pressure does not mean a continuous hypersecretion on the part of the posterior lobe. Functional reflex exhaustion sets in relatively early, and the result is reflex dyspituitarism,* a provision which does away with what would be the necessary result—a continuous hypertension. It is here contended that the symptomatic manifestation of temporary high tension which results from reflex dyspituitarism is headache. It seems further not unfair to infer that the hypophysis responds reflexly not only to changes in pressure upon the cortical cells, but also upon the retinal ganglion cells, a part of the brain substance also subject to variations in pressure.

What part does the ciliary muscle play in the production of a functional disarrangement of the hypophysis? When we compare the eye with the brain, what do we find? Both have an envelope within which the maintenance of a normal tension is one of the requisites for the perfect functioning of its contained nervous structures. The elaborate precautions taken by nature to secure a tension which normally varies within almost inconceivably narrow limits is perhaps exceeded nowhere in the human economy save in the case of the auditory and vestibular terminals to the inner ear. The exquisite delicacy of the vestibular apparatus is probably due to the fact that the instant knowledge of the exact position of our head, without which vertigo ensues, is even more important than sight.

Wegefath (*Journal of Medical Research*, vol. xxxi., 1914-15) has compared in detail the water systems of the cranium and of the eye. He compares the choroid plexuses and the ciliary glands. He calls attention to the epidermal lining of the

ventricles and of the postiridial chambers and to the mesothelial covering of the pia-arachnoid spaces and of the anterior chamber. He compares the arachnoid villi and their relation to the fixed veins with the "pectinate" villi in their relation to the canal of Schlemm. He calls further attention to the fact that "some of the aqueous finds its way backward between the hyaloid and the inner surface of the retina, making its way about the retinal neurons and axons," observations which have an important significance in the physiology of the retina, as well as of the aqueous." It seems not unlikely that the presence of the aqueous, small in amount though it is, between the hyaloid and retina has for one of its purposes the regulation of the pressure on the retinal ganglia in the act of accommodation. The normal variations of pressure on the cerebral cortical ganglia are the result of the activities of the intracranial circulation and the cerebral fluid elaborating organs. On the other hand, the variations of pressure upon the retinal ganglia are not only subject to the normal changes in the intraocular circulation and the amount of aqueous, but to the relatively more violent activities of the ciliary muscle, whose responses to the higher centers for accurate adjustment of the details of the retinal images are often impaired by refractive errors and by lack of tone due to abnormal conditions of the blood circulating through it. Any over or under activity of the ciliary muscle creates a response in the activity of the aqueous secreting glands and results in disturbance of the normal state of the intraocular tension. That in eyes where the excretory system for any reason is imperfect, whether congenitally or from acquired conditions which will ultimately result in glaucoma, strain upon the ciliary muscle from over-use of the eyes in close work results in transient attacks of higher than normal tension, in some cases measurably so. That a relatively higher than normal pressure is exerted upon the retinal ganglion cells in the ordinary case of eyestrain, I think unquestionable, even though the instruments at our command are not delicate enough to measure this rise. In considering the effect of eyestrain, we must bear in mind that we have to deal not only with an over-exerted ciliary muscle but with an ocular water system whose activities are in a measure correlated with those of the ciliary muscle. The

lessening of the activities of this latter as we pass through the various stages of life and the intraocular compensations therefor form an interesting chapter in the history of the eye. In considering intraocular tension, we must bear in mind that we are not dealing with a problem as mechanical as the flow of water through a trough or with a purely capillary action like the absorption of water into a wick, even though these same physical laws play their part. Although the ciliary activities are responded to in the regulation of the intraocular system, they are not necessary to its continuance, as we can see in cases of paralysis of the ciliary muscle. Nor is the response of the ciliary glands merely a transudation which keeps pace with the escape of the aqueous from the eyeball. This is demonstrated in cases of glaucoma. Under normal conditions the regulation of the intraocular tension is under control of a set of complex nervous reflexes. In all likelihood these closely resemble those which regulate the intracranial tension, if indeed they are not part of the same system; and just as in the case of the brain, the normal activities of the choroid plexuses and posterior lobe of the hypophysis vary with the pressure upon the cortical cells, so in the eye the normal activities of the ciliary glands vary with the pressure upon the retinal ganglia.

The eyeball does not seem to provide an agent capable of varying the permeability of the aqueous. I believe, however, that such an agent is necessary to the perfect functioning of the intraocular water system. Less so, however, than the cerebral system perhaps because of the presence of the ciliary muscle whose activities by varying the intraocular pressure in a way unknown in the intracranial system serve as a substitute. That, however, the ciliary muscle is a complete substitute I think most improbable. Wegefarth, as quoted above, says it has been proved possible by varying the pressure relations existing in the cranium and in the eye to demonstrate the direct connection of the two fluid spaces, at least for true solutions. If this be so, then the eye has a means of obtaining from the cerebral fluid a sufficient quantity of posterior lobe secretion to meet the necessities of the aqueous.

If we admit the above facts and contentions, then we can see that eyestrain which results in disturbance of the normal

pressure upon the retinal ganglion cells causes reflexly a disturbance of the secreting activities of the posterior lobe of the hypophysis. If this continues long enough, reflex temporary exhaustion of the posterior lobe activities sets in and with it higher than normal intracranial pressure—the clinical symptom of which is headache. The frequent association of eye-ache after ciliary strain and headache must not be overlooked. Both have the same explanation—hypertension.

Let us examine the symptoms in a few cases of headache.

Mrs. J., aged 47, began to have headaches when she was about thirteen years of age. She has suffered with headaches ever since and they are always worse at time of menstrual flow. Her refractive error is less than one dioptric of farsightedness. There is no astigmatism and no external muscular imbalance. While the use of glasses may have somewhat lessened at times the severity of the headaches, their use did not prevent and seemed in no way to be able to lessen the severity of those of monthly recurrence. In this case the headaches for a short time preceded the full establishment of womanhood and recur with each act of menstruation to the time of menopause. They occur frequently between the menstrual periods and seem then to be brought on by constipation, digestive upsets, physical exhaustion, or strain upon the emotions. The fact, however, that is uppermost in the woman's mind as the result of years of suffering is that the headaches always are more severe at the time of menstruation.

Miss M. B., aged 30, refractive errors none, external muscular conditions normal, a shrivelled, dried-up old woman, "a martyr" her friends all say "to headache." She has never menstruated.

Miss A. J., aged 26, suffers intensely with recurrent headaches and at the menstrual period she is unable to read save with pain; her eyes ache and the sunlight is unbearable. Her refraction and the external ocular conditions are normal.

Miss B., aged 18. Severe recurrent headaches, which are associated with photophobia, pain in the eyeballs and inability to use her eyes, always worse at the time of her menstrual flow, which is accompanied by abdominal pains and pains in the back and nervous symptoms. Refractive and muscular conditions normal.



But why multiply examples? Thousands of similar cases visit the ophthalmologists' offices every month of the year. Question these cases and they tell you of "painful or irregular menstruation," of suppression or absence or irregularity of the menstrual flow, and practically always that their headaches are worse at the time of the menstrual periods. Question their physicians and you get the stories of ante- or retro-flexion, of stenosis, of ovarian cysts, of mal-developments and all the other pictures in the gynecologist's text-books. The result is that ophthalmologists are forced to the belief that there is more than a fancied connection between disturbed ovarian activity and headache. What is it?

None of these cases are as simple as they would appear at first glance. Besides the ovarian abnormalities there will usually be found a history of constipation, and often symptoms which are referable to some chronic focus of infection. How far disturbances of the ovarian secretion has an influence on the activities of the hypophysis and on which lobe cannot be accurately stated. That there is an interrelation of activities is most probable. The disturbance of this interrelationship may have effect in the functioning of the hypophysis in so far as it regulates the intracranial tension and in so far as its influence as explained above produces headache. The concomitant diseases, constipation, digestive disturbances, uterine abnormalities, foci of infection wherever situated, etc., have their effect indirectly upon the ciliary muscle, through changes in the composition of the blood whereby the ciliary effort in accommodation is increased and the difficulties of maintaining normal intraocular conditions are also correspondingly increased. In a similar manner, too, are to be explained the headaches of fever. Every oculist has wondered that so many young people with relatively small errors of refraction "have so much trouble with their eyes," have headache so often following their use and seem to need the assistance of a weak glass. The explanation of this with many cases is that they are suffering from chronic infection of some portion of the exposed lymphatics of the upper air tract, especially of the faucial and post-nasal regions.

Thus we have an explanation why Mrs. Brown needs no glasses, why for Mrs. Smith the use of glasses prevents

headache, and why Mrs. Jones can get little or no relief from their use. In one case, nothing interferes with the normal functioning of the ciliary muscle. There is no disturbance of the intraocular tension and hence no abnormal pressure on the retinal cells. In the second and third cases in different degrees, there are, as the result of blood changes or intraocular abnormalities of development, difficulties in the way of the ciliary muscle continually performing its task without there resulting indirectly undue pressure on the retinal cells, the reflex from which is disturbance of the hypophyseal secretion and, as explained above, headache. We must bear in mind that the retinal cells are as sensitive to changes of pressure as are the cells of the brain cortex and any variation from the normal pressure upon them registers a call for recognition. In the case of the cortical cells, this is taken note of by the intracranial water system. In the case of the retinal cells, there is first an effort of the intraocular water system to give the relief, but where the disturbance is the result of a continuing muscular effort (the ciliary) which is insufficient for the demands made upon it, and where these efforts themselves bring about difficulties in the normal manufacture and escape of the intraocular fluids, there result, on the part of the eye, "pain in the ball," "light shyness," and reflexly, as described above, headache.

In considering headaches, the result of errors of refraction, we must remember that these latter are in any given case constantly present, and that the eye is during the waking hours never altogether free from the "strain" they impose. It is this unremitting over-effort, however varying and slight it may be, that eventually brings about unstable intraocular tension and then reflexly unstable intracranial pressure, which so readily, when the eyes are called upon for excessive effort, result in headache.

This paper would be incomplete without some short comment on a class of cases which has been always, and is to-day, the despair of the ophthalmologists.

Mrs. L. B., aged 41, may be taken for an example. She has had headaches ever since she could remember. It is a headache from which partial surcease has been obtained

for only very short intervals. She has consulted many doctors and submitted with hope and patience to the wearing of many strange medical garments. She has consulted the neurologist, the abdominalist, the internist, the dentist, the surgeon, and the oculist; she has wandered along all the devious paths which lead off from the straight road of medicine, and the headaches continue. Every medical man she has consulted has felt that he has met with something different from the more or less transient headaches with which he has to deal every day, and he undertakes to treat the local trouble, whatever it may be, with a feeling that its relief will in no wise lessen the headache. What is the matter with Mrs. B.? She has what may be termed a "wet" brain, the result of an unrelieved, constant, though varying, high intracranial tension. If I may borrow from ophthalmology a term, I should say Mrs. B. has cerebral glaucoma. The only treatment for them is decompression, but in advising it it should be borne in mind that the operative measures resorted to in the treatment of ocular glaucoma never leave an eye in its first perfect condition and that all such measures are to a great degree only palliative. They, however, often accomplish a great good, and I believe the same may be said of the decompressions done for headache in the cases of "wet" brain.

The case with which "eyestrain" results in a physiological high pressure on the retinal terminals varies with the development of the eyeball. Where the eyeball is small and the lens is large, and where the spaces of Fontana are not amply developed, it will be seen at once that relatively little cilio-crystalline activity will result in a derangement of the physiological intraocular pressure, the pressure on the retinal element will be increased. Reflexly the intracranial pressure will increase. Headache results and with it enforced rest of the cilio-crystalline system. It looks as though the physiological headache had for one of its objects to bring about this result.

The following two observations are of interest here: "Doctor, in the mornings my head aches terribly and I void frequently large quantities of perfectly clear urine. In the afternoon my urine is normal in color, there is very little of it, and my head ceases aching." The patient was forty-odd years of age; his systolic blood pressure 210; advanced nephritis. Mr. L., aged 50, has noticed from time to time

since he was 20 that his headaches would be accompanied by the excretion of large quantities of "urine as clear as water." This "urinary irritation" would cease with the disappearance of the headache. Furthermore he has learned to associate these attacks with prolonged use of the eyes at night and has therefore practically given up reading after dark. It is more than probable that the headache and the kidney disturbance are in both cases secondary to functional exhaustion of the posterior lobe of the hypophysis.



ILLUSTRATING DOCTOR WOODS'S ARTICLE ON "OCULAR ANAPHYLAXIS."



FIG. 1.

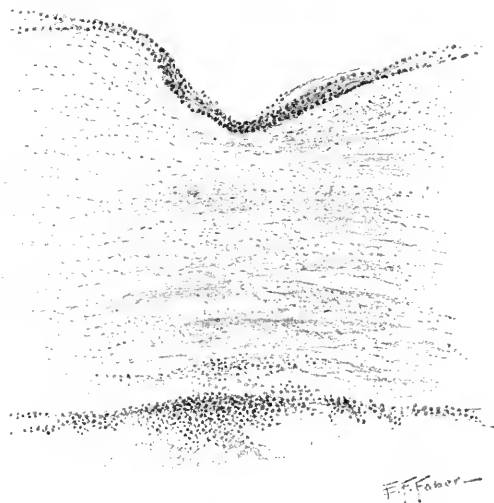


FIG. 3.

## OCULAR ANAPHYLAXIS.

### V. EXPERIMENTAL IRIDOCYCLITIS.

BY DR. ALAN C. WOODS, PHILADELPHIA.

*(With three drawings on Text-Plates IX. and X.)*

(From the John Herr Musser Department of Research Medicine, University of Pennsylvania, Philadelphia.)

**I**N the previous papers (1) of this series, we have presented the evidence, theoretical and technical, which establishes a scientific foundation for the anaphylactic theory of sympathetic ophthalmia. In another paper (2) this evidence has been summed up, and one instance of apparent sympathetic ophthalmia in a dog, and presumably anaphylactic in character, is reported. It is the purpose of this paper to present further observations on the production of anaphylactic iridocyclitis, which apparently represents sympathetic ophthalmia in the dog.

#### TECHNIQUE.

The method employed has been to produce a hypersensitiveness of one eye by means of intraocular injection of homologous (dog's) uveal emulsion in the fellow eye. That such an ocular hypersensitiveness results from the injecting of uveal tissue in the fellow eye has already been demonstrated (1, 2). After a suitable interval, from two to three weeks, in which period sensitization of the second eye occurs, an "intoxicating" intraperitoneal injection of uveal emulsion has been given. Following Elschcnig's observation (3) that sympathetic ophthalmia more frequently occurs in patients showing some other

underlying condition which lowers the natural resistance to disease, we have produced experimentally in several of these animals, morbid conditions of a non-infectious character, chiefly a uranium nephritis, or a phloridzin glycosuria.

Eleven dogs in all have been used in this section of our work. These dogs were carefully selected and were apparently free from all general disease. Before the primary intraocular injection, the eyes of all the animals were carefully examined to insure absence of ocular lesions.

#### CLINICAL MANIFESTATIONS.

After the primary intraocular injection, one of the dogs developed an infection in the injected eye. This dog was at once discarded, as introducing a complicating bacterial element. The remaining ten dogs were under observation for a period of over two months. The injected eyes of these dogs reacted with a more or less severe iridocyclitis which persisted during the period the dogs were under observation.

From within two to three weeks after the intraocular injection of uveal emulsion, the second, or uninjected, eye of these dogs began to show slight symptoms of irritation. These symptoms consisted in a moderate pericorneal injection, together with more or less pronounced photophobia. In those dogs which did not develop an outspoken iridocyclitis, these symptoms and signs of ciliary irritation in the second eye persisted for a period of a week or more, and then gradually cleared up; the eye becoming practically normal.

The "intoxicating" intraperitoneal injection was given from two to three weeks after the primary injection. In the dogs showing an anaphylactic iridocyclitis, an intensification of the symptoms in both the primary injected eye (an iridocyclitis) and in the second eye (symptoms of ciliary irritation) began to develop from within forty-eight hours to a week. In the injected eye the iridocyclitis present since injection became more severe. In the uninjected, or sympathizing eye, the symptoms of ciliary irritation became intensified, the pericorneal injection became more intense, the photophobia more marked, the pupil contracted practically to pin-point size, and then redilated slightly. The iris finally became immobile, slight exudates appeared in the anterior chamber, the edges



ILLUSTRATING DOCTOR WOODS'S ARTICLE ON "OCULAR ANAPHYLAXIS."

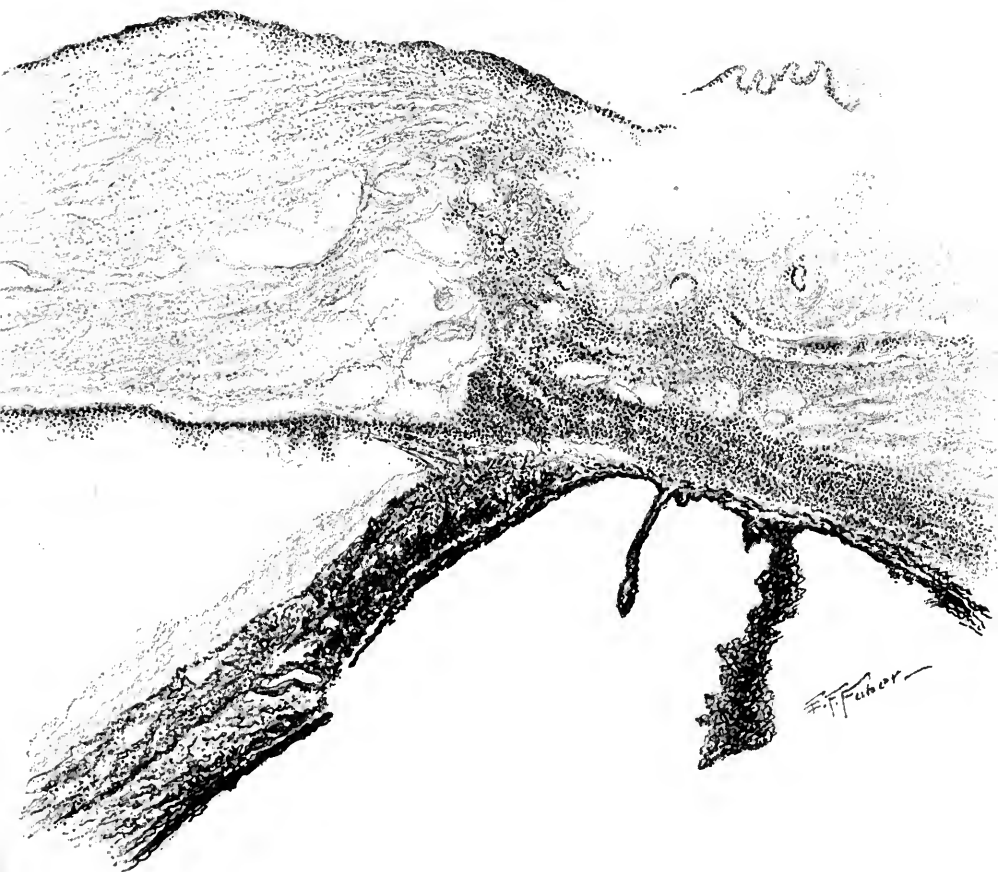


FIG. 2.



of the cornea became dull and grayish, vitreous opacities developed, and the tension of the globe became distinctly lowered. No definite fundus lesions were observed.

Of the ten dogs, one died from a uranium nephritis eight days after receiving the intoxicating dose. Three dogs developed outspoken disease in the second eye, which we believe to be sympathetic ophthalmia. The remaining six dogs developed symptoms of ciliary irritation in the second eye, which persisted for several weeks, with a gradual return to normal.

Of the three dogs developing the anaphylactic iridocyclitis, one has already been reported (2). This dog developed marked symptoms in the second eye within forty-eight hours after receiving the intoxicating dose. These symptoms consisted in pericorneal injection, photophobia, inactive pupil, exudation into the anterior chamber, vitreous opacities, and a diminution in intraocular tension. These symptoms grew rapidly more intense up to the death of the dog ten days later from an intercurrent lobar pneumonia. Histologically this eye showed a round-cell infiltration in the choroidal stroma, especially in the ciliary region, with a slight monocellular infiltration around the ciliary processes (Fig. 1).

The clinical histories of the remaining two dogs are as follows:

*Dog 17-45.* This is an apparently normal dog, whose eyes are entirely negative to external and ophthalmoscopic examination.

*March 16.* Under ether anæsthesia, the anterior chamber of the right eye was tapped and 1.0cc of dog's uveal emulsion was injected into the vitreous of this eye, by injection through the sclera, well back of the scleral-corneal margin.

*March 24.* The dog shows a very slight iridocyclitis of the right eye. The left eye is entirely clear.

*March 30.* The right eye shows a very slight iridocyclitis; the left eye is clear.

*April 4.* The condition of the right eye is unchanged; the left eye remains clear.

*April 7.* The right eye is in the same condition. The left eye to-day shows a slight pericorneal injection, the iris reacts and the media is clear. There is one small hemorrhage near the disk. On this date 9.7cc phloridzin solution<sup>1</sup>

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<sup>1</sup> 1cc phloridzin solution equals 0.2gm phloridzin in sodium carbonate solution. 1.0cc per kilo of body weight was given.

was given intravenously in attempt to establish an underlying disturbance, and 8.0cc dog's uveal emulsion given intraperitoneally to incite lesion in opposite eye.

*April 9.* The iridocyclitis present in the right eye is much more intense and the pupil is contracted to pin-point size. The left eye shows a small pupil and moderate pericorneal injection; the media is clear and the fundus negative.

*April 12.* The iridocyclitis is more marked in the right eye, as is also the pericorneal injection in the left eye; otherwise the condition is unchanged.

*April 17.* The process in the right eye is essentially as before. The left eye shows the pupil contracted to pin-point size and the pericorneal injection is quite intense. The small amount of the fundus that can be seen is negative.

*April 20.* The right eye is as described above. The left eye shows a violent pericorneal injection, the pupil is narrowed, the iris does not react to light, and the edges of the cornea are slightly hazy.

*April 23.* The right eye shows the same intense iridocyclitis, with a slight diminution on palpation in intraocular tension. The left eye shows a violent pericorneal injection and the tension is very low, the eye feeling quite soft. The lower part of the cornea is hazy, the iris is immobile and does not react. There is a small exudate over the dependent portions of the iris, vitreous opacities, and the fundus is not clearly seen. Photophobia is marked.

*April 24.* The right eye shows the same picture and is very soft. The left eye is in the same condition as yesterday, showing violent pericorneal injection, contracted and immobile iris, with a faint exudate in the anterior chamber, a hazy cornea, and vitreous opacities.

*April 25.* Both eyes are in the same condition, the intraocular tension is very low, both eyes being soft and mushy. The dog was killed with chloroform, autopsied, and the eyes and specimens of the tissues saved for section.

*Dog 16-51.* This is an apparently normal dog; both eyes are entirely negative to external and ophthalmoscopic examination.

*March 16.* Under ether anæsthesia the anterior chamber of the right eye was tapped, and 1cc of dog's uveal emulsion injected into the posterior chamber in the usual manner.

*March 24.* The right eye shows a slight anterior staphyloma, and a hemorrhagic area surrounding the cornea. There is present the usual evidence of an iridocyclitis. The left eye is clear.

*March 30.* The right eye is unchanged. There is the

faintest suggestion of pericorneal injection in the left eye. Otherwise it appears normal.

*April 4.* Both eyes are in the same condition as when last noted.

*April 7.* The right eye shows an iridocyclitis, with considerable lowering of the intraocular tension. The left eye to-day shows a moderate pericorneal injection. The iris appears normal, the media is clear, and the fundus negative.

*8cc dog's uveal emulsion given intraperitoneally.*

*10.7cc phloridzin solution given intravenously.*

*April 9.* The eyes appear in practically the same condition. The pericorneal injection in the left eye is possibly a little more intense.

*April 12.* The eyes continue in the same condition.

*April 17.* The right eye shows an intense iridocyclitis, moderate anterior staphyloma, and a diminution in intraocular tension. The left eye to-day shows a deep and intense pericorneal injection, the pupil is somewhat contracted, the media is clear, and the fundus appears normal. There is a definite photophobia.

*April 20.* The right eye is as described in last note but in the left the pericorneal injection and photophobia are both somewhat more marked.

*April 23.* The right eye is unchanged, but the left eye shows violent pericorneal injection, a contracted pupil, and the iris does not react to light. The edges of the cornea are slightly hazy, the media clear, and the fundus negative.

*April 24.* The right eye is unchanged. The pupil of the left eye is contracted to pin-point size and the condition described yesterday persists unabated.

*April 25.* The right eye shows a cloudy cornea with anterior staphyloma, an intense iridocyclitis, and a great diminution in intraocular tension. The left eye shows violent pericorneal injection, a hazy cornea, an inactive, lusterless iris, slight exudates in the anterior chamber, and vitreous opacities. The fundus is not clearly seen and the tension is distinctly lower. The photophobia is marked.

*April 27.* The condition of both eyes is essentially unchanged except that the intraocular tension appears lower. The dog was killed with chloroform, autopsied, and the eyes and specimens of the tissues preserved for section.

#### PATHOLOGY.

##### 1. General.

*17-45.* At autopsy this dog showed a typical distemper broncho-pneumonia, characterized by the presence in the right lung of several discrete, elevated areas about 1.5cm

in diameter. Section through such areas showed a pneumonic process in the stage of red hepatization, extending out from the bronchus. The lower lobe of the left lung showed a larger pneumonic process involving half of the lobe. On section this appeared to be a fused broncho-pneumonia in the stage of red hepatization. All other tissues appeared normal.

The histological examination of the tissues of this dog showed a typical broncho-pneumonia in the right lung and the lower lobe of the left lung. In both kidneys there was a very slight pyelonephritis characterized by focal collections of mononuclear and polynuclear leucocytes along the papillary ducts in the pelvis. The other tissues all appeared normal.

16-51. This animal showed no evidence of any gross or histological change in any organ, except the eye.

## 2. *Special (Ocular).*

*Gross.* The gross appearance of the eyes of the two dogs was essentially the same. The right eye, which had been injected in each instance with uveal emulsion and which presumably acted as the exciting eye, had the following gross appearance. The cornea appeared normal. There was a faint fibrinous exudate in the anterior chamber and both the iris and the ciliary body appeared definitely swollen, the former being adherent to the anterior capsule of the lens. Over the ciliary body, extending forward in the posterior chamber and surrounding the lens, there was a web-like fibrinous exudate. The retina and choroid showed no gross lesions.

The gross appearance of the left, or sympathizing, eye was very similar in the two dogs. The cornea appeared to be slightly thickened. There was a slight exudate in the angles of the anterior chamber and in both dogs the iris was apparently thicker than normal. In dog 16-51, the iris was adherent to the anterior capsule of the lens by fibrinous synechiæ. In dog 17-45 synechiæ were not present. The ciliary body did not appear to be swollen but was covered, in both instances, by a fibrinous exudate, which extended forward in the anterior part of the posterior chamber, up to and surrounding the lens. There were no apparent gross retinal or choroidal lesions.

*Microscopic.* The pathological process in the eyes of both dogs was identical but appeared to be slightly more advanced in 17-45. The microscopic appearance of the eyes of this dog is described below.

*Right* (injected eye). The anterior segments of the

choroid, especially those parts underlying the ciliary processes, show a definite flat thickening. This thickening extends forward and involves the iris. Throughout the stroma of this section of the choroid, and the stroma of the iris, there is a moderate cellular infiltration, which appears to be mononuclear in type. In the anterior half of the posterior chamber there is a slight fibrinous exudate extending up to the margins of the lens. In this exudate are enmeshed a very few free pigment granules. The retina does not appear to be involved in the pathological process. The anterior chamber, Descemet's membrane, and cornea appear normal.

*The left eye*, which showed clinically the anaphylactic iridocyclitis, presented the following histological picture (Fig. 2). The anterior segments of the choroid, especially those portions underlying the ciliary processes, showed a marked thickening. The stroma of this section of the choroid is the seat of a dense mononuclear infiltration which extends forward and into the stroma of the iris, which is correspondingly thickened and shows the same cellular infiltration. The anterior endothelial layer of the iris is covered with a faint, homogeneous, eosin-staining exudate. The angles of the anterior chamber are involved in this same process and show a mononuclear exudate, which involves the ciliary body, and, extending forward, involves also the pectinate ligament, the lymph spaces, and Schlemm's canal, in which is a thin sero-fibrinous exudate. At this point, at the angle, there is a direct continuity between the infiltration of the choroid, the iris, and pectinate ligament. There is a moderate involvement of the ciliary processes, which are surrounded by a slight but definite mononuclear and fibrinous exudate.

The cornea is thickened, but the thickening appears due entirely to an interlamellar oedema, without the presence of any cellular elements. Descemet's membrane is, however, definitely involved in a process which is continuous with the involvement of the pectinate ligament. The endothelium shows a definite proliferation, and is covered with a faint, eosin-staining, homogeneous exudate, in which are enmeshed occasional lymphocytes (Fig. 3).

In the cellular infiltration of the stroma of the choroid, the mononuclear cells show occasional slight clumping, suggesting somewhat giant cells, but nowhere are true giant cells found.

The pathological process in the "sympathizing" eye is, in short, a chronic iridocyclitis, with involvement of the pectinate ligament, of the lymph spaces, and Schlemm's canal, and extending also to Descemet's membrane.

## INFLUENCE OF OTHER MORBID CONDITIONS.

Elschnig (5) has commented upon the frequency, if not constancy, with which other pathological conditions, such as nephritis, diabetes, pulmonary diseases, etc., are encountered in patients with sympathetic ophthalmia. This observation has led him to suggest that such conditions may be predisposing factors in the development of sympathetic ophthalmia, after injury or disease of the exciting eye, inasmuch as such diseases lower the general resistance and make the organism more subject to anaphylactic reactions.

Following this suggestion, several of the experimental dogs have been given subcutaneous injections of uranium nitrate, and intravenous injections of phloridzin, thus producing an acute nephritis or a phloridzin glycosuria. Two dogs with uranium nephritis died in ten days and one month respectively. The dog dying in ten days showed no symptoms of any kind in the second eye. The dog dying after a month showed a very slight sympathetic irritation which appeared about three weeks after the injection of uveal emulsion in the exciting eye and persisted without any increase in intensity up to the dog's death ten days later.

Three dogs were given injections of phloridzin. Two of these were the dogs showing the anaphylactic iridocyclitis.

Of more interest, however, were the pulmonary lesions in the dogs showing the experimental irido-cyclitis. One of the dogs showed a definite lobar pneumonia in the stage of gray hepatization. The cause of this pneumonia was not determined. Another dog showed what was apparently a typical distemper broncho-pneumonia. The lungs of the third dog were apparently normal. This pulmonary condition seems especially interesting, inasmuch as it is well known that an anaphylactic reaction produces a definite pulmonary condition. It seems possible, although it is only a conjecture, that the resistance of the lungs to infection may have thus been lowered by the anaphylactic condition.

The second possibility is that the pneumonia was the primary process and that the ocular condition was secondary, due to bacterial metastasis. This possibility seems unlikely for the following reasons. The ocular condition in the three



dogs appeared clinically and histologically to be the same morbid process. Two of the dogs showed a pneumonia, the third dog appeared at autopsy to be entirely normal. The histological picture shown by the eyes of these dogs was in no sense the picture of a bacterial infection. The ocular process, although violent, leading to practical loss of the ball, involved only the uveal tract, and did not extend to any of the other tissues of the eye, which is not the course of a metastatic bacterial infection of the eye. Moreover, if the ocular condition were due to bacterial metastasis, there should be evidence of such metastasis in other organs. One dog, 17-45, showed, it is true, a slight pyelonephritis, but this in turn was probably secondary to the phloridzin injection. The remaining dogs showed no evidence of any bacterial metastasis. For these reasons, it appears probable to us that the ocular lesions were totally independent of the pulmonary lesions.

The three instances of anaphylactic iridocyclitis, apparently sympathetic in character, which are reported, occurred, therefore, either in dogs subjected to a phloridzin glycosuria, or in dogs which, during the period of observation, developed pneumonia. Five dogs, normal except for the sensitizing and intoxicating injections of uveal emulsion, developed no symptoms in the second eye, beyond the symptoms of a more or less mild sympathetic irritation. It seems probable, therefore, that Elschnig's suggestion that some other anomaly of the organism is needed to decrease the resistance, and so allow the development of sympathetic ophthalmia, is a correct view.

#### DISCUSSION.

The pathology of sympathetic ophthalmia has been the subject of much investigation, and it is generally conceded that the picture is fairly characteristic. According to Uhr (4), Ruge (5), and Fuchs (6) sympathetic ophthalmia is manifested pathologically as a chronic fibrino-plastic uveitis. The pathological changes are limited entirely to the uvea. Uhr describes the picture as characterized by a thickening of the choroid due to a monocellular infiltration. There is a predilection of this infiltration for the external layers of the

suprachoroid and occasionally a progression of the infiltration into the sclera, along the vessel walls, and along the lymph spaces of the ciliary nerves,—a process somewhat similar to a perivascular lymphangitis. Among the mononuclear cells are numerous mast cells, with epithelioid and giant cells, which may be massed in the infiltrated uvea as in a tubercle. Ruge emphasizes the fact that the disease is limited to the uvea, and is characterized by a monocellular infiltration, partially diffuse and partially circumscript, around the blood-vessels. Epithelioid and giant cells are present in the cellular infiltration and there is also a mononuclear infiltration around the ciliary process. This picture is essentially similar to that described by Fuchs.

In many respects the experimental iridocyclitis we have reported here resembles this histological picture. In other respects it is, however, different. The involvement of the pectinate ligament and Descemet's membrane is manifestly directly dependent upon the process in the iris and choroid, and is but a direct extension of this. The term "anterior uveitis" has indeed well been applied to this apparent corneal condition. In so far as this anaphylactic iridocyclitis is a chronic affair, characterized by a mononuclear infiltration, and limited to the uvea, it resembles the picture of human sympathetic ophthalmia described by Uhr and others. Moreover, the processes are similar in that both tend to involve the lymph spaces.

The processes differ in that the iridocyclitis in the dog involves especially, if not solely, the anterior portion of the uveal tract, while sympathetic ophthalmia in man involves especially the posterior portion of the uvea. However, in spite of this difference, the two conditions appear to us to be the same. The experimental condition appears to be sympathetic, following injury to the fellow eye. The same vital structure is involved in both conditions. The nature of the involvement is essentially the same in both conditions. Finally the difference in species may be sufficient to explain the difference in spite of the involvement in the uvea. To us the most important thing is that the same tissue in the dog and in man is involved in a similar pathological process.

Further minor differences are present, notably the occur-

rence of epithelioid and giant cells in the human disease, and the absence of these cells in the disease here reported. This last difference, however, we are inclined to minimize. It seems probable that the giant cells and epithelioid cells are in no way characteristic of the disease, but are merely an evidence of a greater chronicity, for, as a rule, the occurrence of such elements is but an evidence of chronicity—continued irritation. The resistance of dogs toward a chronic disease, the scarcity of such disease, and the difficulty, if not impossibility of producing chronic disease in dogs, is well known. Experimentally, as a rule, dogs respond to insults with an acute reaction, which speedily leads either to recovery, to loss of the organ involved, or to death, as the case may be. The most reasonable view, therefore, of the absence of epithelioid and giant cells in this condition in the dog and the occurrence of these elements in sympathetic ophthalmia in man, is that the anaphylactic iridocyclitis in the dog is more acute than the analogous process in man.

The early contraction of the pupil shown by these dogs is of especial interest. In the early studies in this series it was shown that when the eyes were perfused with specific antigen the pupil of a sensitized dog responded by a marked contraction. Further it is stated by Nettleship that oscillation of the pupil is one of the earliest manifestations of impending sympathetic ophthalmia.

In reporting this experimental condition it is realized that it is not a typical sympathetic ophthalmia, as the term is used clinically. Working with lower animals, it is almost hopeless to expect a typical reproduction of such a disease. Yet that this condition is sympathetic ophthalmia in the dog, we do believe. It follows injury to the fellow eye where uveal tissue is liberated for absorption, and is produced by an anaphylactic mechanism. The process in the sympathizing eye is limited to the uvea. The underlying pathological nature is similar to that of sympathetic ophthalmia as we recognize it in man.

If our supposition is true, that this anaphylactic iridocyclitis is a true sympathetic ophthalmia in the dog, it is the last step in the experimental demonstration of the anaphylactic nature of sympathetic ophthalmia. We believe the questions of ocular anaphylaxis, the antigenic properties of uveal pigment,

and the experimental reproduction of a sympathetic ocular disease in dogs by anaphylactic means, to be established. Whether or not these purely laboratory and experimental findings can be brought in relationship with clinical sympathetic ophthalmia remains to be seen. We fully appreciate that the final demonstration of the true nature of sympathetic ophthalmia, or indeed of any disease, must be in man.

#### SUMMARY.

An anaphylactic iridocyclitis has been produced in dogs by sensitization through intraocular injections of dog's uveal emulsion in the fellow eye, and intoxication through intraperitoneal injection. The condition begins with signs of ciliary irritation, followed, in such dogs as develop the disease, by an outspoken iridocyclitis, with subsequent descemetitis. Three out of ten animals developed such a condition. Histologically the process is a thickening and round-cell infiltration of the anterior portion of the choroid, and the iris, with an involvement along the pectinate ligament, of Schlemm's canal, and a descemetitis. From the mode of occurrence, character, and pathology of the disorder, it is believed to be sympathetic ophthalmia in the dog.

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## THE SURGICAL TREATMENT OF CORNEAL SUP- PURATION IN EXOPHTHALMIC GOITER.<sup>1</sup>

BY DR. ARNOLD KNAPP, NEW YORK.

**S**LIGHT corneal involvement in exophthalmic goiter is easily controlled by care and simple remedies. The severer corneal lesions, particularly when they occur in the toxic cases, are desperate and tax the surgeon's resources to the utmost. The loss of both corneæ in exophthalmic goiter is not so very unusual; Sattler, in 1907, collected the reports of forty cases where both eyes were lost. The following case of exophthalmic goiter where one eye was lost and the other eye was saved with some vision may be of interest.

*B. K.*, aged 35, stated that his eyes began to become prominent in August, 1916. His general condition during the past six months has been very much affected, and he has lost forty pounds in weight. On Jan. 13, 1917, the patient came to the Herman Knapp Memorial Eye Hospital on account of pain in the left eye. Both eyes were very prominent; the right eye could be closed only with effort, and at night this eye was kept open; there was some conjunctival congestion. The conjunctiva of the left eye was red and chemosed, the cornea was infiltrated with pus and was dried out, a little clear cornea remained in the upper part. Vision: R = 20/30; L = 1/200.

The patient was restless, anxious, with occasional tremor and a dry skin. Pulse about 90. The thyroid gland was hard, fibrous, but not enlarged. No difficulty on swallowing. He had a heart lesion and weighed 115 pounds. Dr. John Rogers of New York was kind enough to see the patient and prescribed adrenalin and advised the ligation of the

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<sup>1</sup> Read at meeting of American Academy of Ophthalmology and Otolaryngology at Pittsburg, October 29-30, 1917.

left inferior thyroid artery. This operation was done. During the next few days his general condition remained about the same; at times he was alert and quiet, at other times he was restless, apathetic and could not sleep at night, and had no appetite. He had no diarrhoea. The congestion of the right eye was relieved and the cornea remained intact. The process in the left eye continued and the cornea sloughed. Jan. 20th: blood-pressure 104-110; weight 110½ lbs. As the patient was losing weight steadily, Dr. Rogers advised transferring him to the St. Francis Hospital, where he was better prepared to ligate the remaining thyroid arteries, which was then done.

On Feb. 5th the right eye had again become inflamed and a part of the outer wall of the orbit was resected by Dr. Rogers to relieve the intraorbital pressure. I saw the patient on Feb. 11th, as the right eye was not doing well. I found the right conjunctiva chemosed and swollen and the cornea in its lower half infiltrated with pus. The patient was immediately returned to the eye hospital and an attempt was made to arrest the process in the right cornea by frequent irrigation, applications of argyrol salve, and bandaging. On Feb. 12th the corneal condition was worse: the part of the cornea above, which had remained clear, had now become clouded. There was very little discharge. The general condition of the patient was about the same, except that he had lost ten additional pounds.

Under ether it was decided to suture the eyelids, following out the suggestion of Priestly Smith mentioned in the discussion of Juler's article<sup>1</sup> in the *T. O. S.*, 1913. An intermarginal incision was made in both lids, splitting the lids rather deeply. Mattress sutures were then introduced from above through the cut part of the lids, drawing together the bottoms of the two grooves, and the lids were approximated without much tension throughout a broad contact of the cut surfaces. Cutaneous incisions were made along the margins of the orbit above and below and on the temporal side in order to relieve the tension of the skin, as suggested by Bishop Harmon.<sup>2</sup> The left eye was eviscerated. At the following daily dressings the lids of the red eye looked normal, though somewhat bulging, and the sutures were apparently holding. The conjunctival sac was irrigated daily from the sides. The patient's general condition was about the same. A small opening formed at the center of the lids through which the progress of the corneal lesion could be watched. The upper part of the cornea cleared, a

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<sup>1</sup> Juler, F. A., "Acute Purulent Keratitis in Exophthalmic Goiter," *T. O. S.*, 1913, p. 71.

<sup>2</sup> *L. c.*, p. 74.

small perforation took place below, and a necrotic plug was cast off. On Feb. 20th his weight was 91½ lbs. On the fifth day the sutures were removed on account of stitch-infection, the lids however remained united. The patient was put on thyroid residue in addition to the suprarenal extract, and his general condition began to improve. The upper third of the cornea remained clear. Several decayed teeth were extracted, and the general improvement continued. The points of union of the lids slowly stretched, forming bands 2-3mm long, the cornea nevertheless was sufficiently protected. The general health showed steady improvement, but there was no recession of the exophthalmus. The bands uniting the lids were divided and this fall an optical iridectomy was performed upward; the outer third of the palpebral fissure was sutured to facilitate closing the lids. The vision was 6/200.

This desperate case is instructive from a number of points of view. It shows first that there is no general treatment which will lessen the exophthalmus promptly enough to safeguard the affected cornea. C. H. Mayo and others recommend the resection of the superior sympathetic ganglion under these conditions. This procedure probably offers the most hope though the operation is not simple, and our patient's poor general condition did not warrant so severe a surgical intervention. Juler (*l. c.*) has investigated the results of sympathetomy, and states that Balacescu<sup>1</sup> found that after operation out of 41 cases the exophthalmus was improved in 34 after a few hours or days, in 3 cases one eye only was improved, in 4 there was no effect. Later results were given in 25 cases, and marked improvement and even disappearance of the symptoms were observed in 19, no change in 3, increase in 3. In Juler's own case no marked recession of the exophthalmus occurred after this operation. In other words, favorable influence on the exophthalmus is by no means obtained in all cases. At the same time, in view of the desperate nature of the condition, if the patient's condition permits, this operation should be attempted.

The local condition remains the important one to correct, and if this can be relieved the corneal process will be arrested. The important contributing cause is the chemosed and thick-

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<sup>1</sup> Balacescu, *Arch. f. klin. Chirurgie*, 1912.

ened conjunctiva in the lower half of the eyeball, which mechanically prevents the closure of the lids. The lids must be brought together to cover the cornea. Suture of the lids has undoubtedly been practiced by everyone for this purpose, but ordinary suturing even with freshening of the lid-margins is not satisfactory, as the sutures do not hold. In my opinion, the suture method of Priestly Smith, augmented by the releasing cutaneous incisions of Bishop Harmon, meets these conditions best, and the favorable result obtained in this case must be ascribed to it.



## NARROWING OF THE PUPIL DOES NOT LOWER NORMAL INTRAOCULAR TENSION (100 CASES).<sup>1</sup>

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THESE observations were made to determine whether or not there is a lowering of normal intraocular tension when the pupils are made smaller. The necessity for determining this matter arose from our routine clinical work on three types of cases.

1st. "Borderline Glaucoma" cases.

In these cases we were dealing with slightly enlarged pupils, suspicious cups, inconclusive narrowing of fields, and a tension above, say, 25mm Hg. We often noted a definite lowering of tension after a miotic, while at other times there was no change.

2d. Definite "high tension" simple glaucoma in one, with tension well within so-called normal limits in the other eye. The tension frequently fell in this second eye though in some cases it remained unchanged after miosis (Storey).

3d. Acute inflammatory glaucoma in one eye with normal tension in the other eye unaffected by narrowing of the pupil (Field).

It is at once evident that in these types of cases it is of prime importance to know whether or not normal tension is reducible by miotics.

The material consists of 100 cases studied at the Cook

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<sup>1</sup> From the Research Laboratories of Cook County Hospital and Eye Clinic of Dr. E. V. L. Brown.

Read before the Chicago Ophthalmological Society, November, 1917.

County Hospital. They were patients that gave no history of eye trouble and were not seriously sick. Their ages ranged from 12 to 67 years. Only cases in which satisfactory tonometer readings could be made were used.

As in a previous series of cases,<sup>1</sup> all tension readings were taken with the Schiotz tonometer, and holocain in 1% sol. was used as an anæsthetic. The size of the pupil was first noted—then the tension taken. One half hour later the tension was again taken. The pupils were then rather quickly contracted with eserine salicylate in 1 or 2% sol. The average size of the pupils after the miosis was  $1\frac{1}{4}mm$ . All pupils were contracted to at least 2mm. Holocain was again instilled and the final tonometric readings taken.

*In no case was there a lowering of tension following the contraction of the pupil greater than 3mm Hg.*

This amount is well within the recognized limits of error of observation in the use of the tonometer. There was in no case any difference between the first and second readings before the miosis.

Following the use of the eserine many patients of course complained of diminution of vision and headache; a few of pain within the eyes; a few became nauseated; while four or five vomited. The pupils remained contracted a variable length of time—in a few eyes as long as seventy-two hours.

From this series of 100 cases we may therefore conclude that: intraocular tension in normal eyes is not to be lowered by the narrowing of the pupil. The corollary of this statement, namely, that a decrease of intraocular pressure following the use of a miotic necessarily means that an eye is glaucomatous, is not even herewith postulated and does not properly come within the scope of this paper. We can merely verify the statement frequently made—Narrowing the pupil does not lower normal intraocular tension.

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<sup>1</sup> "The Effect of Tonsillectomy on Normal Intraocular Tension" (100 cases), ARCH. OF OPHTH., vol. xlvii., No. 1, p. 46.

TABLE.

CASE No.	AGE.	BEFORE MIOSIS.						AFTER MIOSIS.			
		PUPILS.		TENSION.				PUPILS.		TENSION.	
				First.		$\frac{1}{2}$ hour later.					
		R. mm	L. mm	R. mm	L. mm	R. mm	L. mm	R. mm	L. mm	R. mm	L. mm
1	27	4	4	14 $\frac{1}{4}$	13 $\frac{1}{4}$	14 $\frac{1}{4}$	12 $\frac{3}{4}$	1	1	15 $\frac{1}{4}$	13 $\frac{1}{4}$
2	44	2 $\frac{1}{2}$	2 $\frac{1}{2}$	12 $\frac{1}{4}$	16 $\frac{1}{2}$	14 $\frac{1}{4}$	16 $\frac{1}{2}$	1	1	13 $\frac{1}{4}$	15 $\frac{1}{4}$
3	39	2 $\frac{1}{2}$	2 $\frac{1}{2}$	16 $\frac{1}{2}$	16 $\frac{1}{2}$	16 $\frac{1}{2}$	16 $\frac{1}{2}$	1	1	18	18
4	25	4	4	13 $\frac{1}{4}$	13 $\frac{1}{4}$	13 $\frac{1}{4}$	14 $\frac{1}{4}$	1 $\frac{1}{4}$	1 $\frac{1}{4}$	13 $\frac{1}{4}$	13 $\frac{1}{4}$
5	21	4	4	18	19 $\frac{1}{2}$	18	19 $\frac{1}{2}$	1 $\frac{1}{2}$	1 $\frac{1}{2}$	19 $\frac{1}{2}$	18
6	23	5	5	18	18	16 $\frac{1}{2}$	15 $\frac{1}{4}$	1 $\frac{1}{4}$	1 $\frac{1}{4}$	18	18
7	30	6	5	12 $\frac{1}{4}$	11 $\frac{1}{4}$	12 $\frac{1}{4}$	13 $\frac{1}{4}$	1	1	14 $\frac{1}{4}$	14 $\frac{1}{4}$
8	31	3 $\frac{1}{2}$	3 $\frac{1}{2}$	19 $\frac{1}{2}$	19 $\frac{1}{2}$	21 $\frac{1}{2}$	18	1 $\frac{1}{4}$	1 $\frac{1}{4}$	19 $\frac{1}{2}$	18
9	36	3	3	16 $\frac{1}{2}$	16 $\frac{1}{2}$	16 $\frac{1}{2}$	15 $\frac{1}{4}$	1	1	18	16 $\frac{1}{2}$
10	57	5	5	16 $\frac{1}{2}$	16 $\frac{1}{2}$	15 $\frac{1}{4}$	16 $\frac{1}{2}$	1 $\frac{1}{4}$	1 $\frac{1}{4}$	15 $\frac{1}{4}$	18
11	56	3 $\frac{1}{2}$	3 $\frac{1}{2}$	13 $\frac{1}{4}$	13 $\frac{1}{4}$	12 $\frac{1}{4}$	13 $\frac{1}{4}$	1 $\frac{1}{2}$	1 $\frac{1}{2}$	13 $\frac{1}{4}$	13 $\frac{1}{4}$
12	72	4	4	18	18	18	21	2	2	18	18
13	39	4	4	15 $\frac{1}{4}$	16 $\frac{1}{2}$	15 $\frac{1}{4}$	15 $\frac{1}{4}$	1	1	15 $\frac{1}{4}$	16 $\frac{1}{2}$
14	37	4	4	13 $\frac{1}{4}$	14 $\frac{1}{4}$	13 $\frac{1}{4}$	14 $\frac{1}{4}$	1 $\frac{1}{2}$	1 $\frac{1}{2}$	13 $\frac{1}{4}$	15 $\frac{1}{4}$
15	41	4	4	18	19 $\frac{1}{2}$	18	19 $\frac{1}{2}$	1 $\frac{1}{2}$	1 $\frac{1}{2}$	19 $\frac{1}{2}$	19 $\frac{1}{2}$
16	65	5	5	21	23	23	23	1 $\frac{1}{2}$	1 $\frac{1}{2}$	21	21
17	61	4	5	14 $\frac{1}{4}$	14 $\frac{1}{4}$	14 $\frac{1}{4}$	14 $\frac{1}{4}$	1	1	14 $\frac{1}{4}$	13 $\frac{1}{4}$
18	35	5	5	14 $\frac{1}{4}$	14 $\frac{1}{4}$	13 $\frac{1}{4}$	14 $\frac{1}{4}$	1 $\frac{1}{2}$	1 $\frac{1}{2}$	14 $\frac{1}{4}$	14 $\frac{1}{4}$
19	49	5	5	14 $\frac{1}{4}$	14 $\frac{1}{4}$	14 $\frac{1}{4}$	14 $\frac{1}{4}$	1	1 $\frac{1}{4}$	14 $\frac{1}{4}$	15 $\frac{1}{4}$
20	25	4	4	12 $\frac{1}{4}$	12 $\frac{1}{4}$	12 $\frac{1}{4}$	13 $\frac{1}{4}$	1	1	12 $\frac{1}{4}$	14 $\frac{1}{4}$
21	21	4 $\frac{1}{2}$	4 $\frac{1}{2}$	13 $\frac{1}{4}$	14 $\frac{1}{4}$	13 $\frac{1}{4}$	14 $\frac{1}{4}$	1	1	11 $\frac{1}{2}$	11 $\frac{1}{2}$
22	24	4	4	14 $\frac{1}{4}$	16 $\frac{1}{2}$	15 $\frac{1}{4}$	15 $\frac{1}{4}$	1	1	13 $\frac{1}{4}$	13 $\frac{1}{4}$
23	15	3 $\frac{1}{2}$	3 $\frac{1}{2}$	13 $\frac{1}{4}$	13 $\frac{1}{4}$	13 $\frac{1}{4}$	13 $\frac{1}{4}$	1	1	13 $\frac{1}{4}$	12 $\frac{1}{4}$
24	14	3	3	19 $\frac{1}{2}$	19 $\frac{1}{2}$	19 $\frac{1}{2}$	19 $\frac{1}{2}$	2	1 $\frac{1}{2}$	19 $\frac{1}{2}$	18
25	43	3	3	16 $\frac{1}{2}$	16 $\frac{1}{2}$	16 $\frac{1}{2}$	15 $\frac{1}{4}$	1	1 $\frac{1}{4}$	14 $\frac{1}{4}$	15 $\frac{1}{4}$
26	38	4	4	13 $\frac{1}{4}$	13 $\frac{1}{4}$	14 $\frac{1}{4}$	12 $\frac{1}{4}$	1 $\frac{1}{2}$	1 $\frac{1}{2}$	14 $\frac{1}{4}$	15 $\frac{1}{4}$
27	28	4	4	12 $\frac{1}{4}$	13 $\frac{1}{4}$	13 $\frac{1}{4}$	12 $\frac{1}{4}$	$\frac{3}{4}$	$\frac{3}{4}$	13 $\frac{1}{4}$	13 $\frac{1}{4}$
28	21	4 $\frac{1}{2}$	4 $\frac{1}{2}$	18	18	18	16 $\frac{1}{2}$	1 $\frac{1}{4}$	1 $\frac{1}{4}$	19 $\frac{1}{2}$	18
29	53	3	3	18	16 $\frac{1}{2}$	16 $\frac{1}{2}$	16 $\frac{1}{2}$	1	2	18	19 $\frac{1}{2}$
30	23	3 $\frac{1}{2}$	3 $\frac{1}{2}$	15 $\frac{1}{4}$	16 $\frac{1}{2}$	18	19 $\frac{1}{2}$	1 $\frac{1}{4}$	1	18	16 $\frac{1}{2}$
31	55	4 $\frac{1}{2}$	4 $\frac{1}{2}$	16 $\frac{1}{2}$	16 $\frac{1}{2}$	14 $\frac{1}{4}$	16 $\frac{1}{2}$	1 $\frac{1}{2}$	1 $\frac{1}{2}$	13 $\frac{1}{4}$	14 $\frac{1}{4}$
32	19	3 $\frac{1}{2}$	3 $\frac{1}{2}$	16 $\frac{1}{2}$	15 $\frac{1}{4}$	18	15 $\frac{1}{4}$	1 $\frac{1}{2}$	1 $\frac{1}{2}$	18	18
33	51	4	3	15 $\frac{1}{4}$	16 $\frac{1}{2}$	15 $\frac{1}{4}$	16 $\frac{1}{2}$	1	1	14 $\frac{1}{4}$	15 $\frac{1}{4}$
34	76	4 $\frac{1}{2}$	4 $\frac{1}{2}$	21	21	21	19 $\frac{1}{2}$	1 $\frac{1}{2}$	1 $\frac{1}{2}$	21	18
35	49	4	3 $\frac{1}{2}$	16 $\frac{1}{2}$	16 $\frac{1}{2}$	16 $\frac{1}{2}$	14 $\frac{1}{4}$	1 $\frac{1}{2}$	1	16 $\frac{1}{2}$	13 $\frac{1}{4}$
36	12	4	4	15 $\frac{1}{4}$	14 $\frac{1}{4}$	13 $\frac{1}{4}$	16 $\frac{1}{2}$	1 $\frac{1}{2}$	1 $\frac{1}{2}$	14 $\frac{1}{4}$	15 $\frac{1}{4}$
37	62	4 $\frac{1}{2}$	4 $\frac{1}{2}$	14 $\frac{1}{4}$	14 $\frac{1}{4}$	16 $\frac{1}{2}$	15 $\frac{1}{4}$	1 $\frac{3}{4}$	1 $\frac{1}{4}$	14 $\frac{1}{4}$	13 $\frac{1}{4}$
38	12	4	4	10	10	10	10	2	2	12 $\frac{1}{4}$	11 $\frac{1}{4}$
39	30	4 $\frac{1}{2}$	5	13 $\frac{1}{4}$	12 $\frac{1}{4}$	14 $\frac{1}{4}$	12 $\frac{1}{4}$	1 $\frac{1}{4}$	1 $\frac{1}{4}$	13 $\frac{1}{4}$	10
40	18	4	4	18	19 $\frac{1}{2}$	18	18	1	1	16 $\frac{1}{2}$	16 $\frac{1}{2}$
41	46	3	3	16 $\frac{1}{2}$	16 $\frac{1}{2}$	18	16 $\frac{1}{2}$	1	1	16 $\frac{1}{2}$	16 $\frac{1}{2}$
42	48	4	4	18	16 $\frac{1}{2}$	18	18	1	1	18	18
43	64	4	4	14 $\frac{1}{4}$	15 $\frac{1}{4}$	15 $\frac{1}{4}$	15 $\frac{1}{4}$	1	1	14 $\frac{1}{4}$	14 $\frac{1}{4}$
44	39	3	3	15 $\frac{1}{4}$	15 $\frac{1}{4}$	15 $\frac{1}{4}$	15 $\frac{1}{4}$	1 $\frac{1}{2}$	1 $\frac{1}{2}$	14 $\frac{1}{4}$	15 $\frac{1}{4}$
45	35	5	5	18	18	18	18	1 $\frac{1}{2}$	1 $\frac{1}{2}$	18	18

CASE No.	AGE.	BEFORE MIOSIS.						AFTER MIOSIS.					
		PUPILS.		TENSION.				PUPILS.		TENSION.			
				First.		$\frac{1}{2}$ hour later.							
R.	L.	R.	L.	R.	L.	R.	L.	R.	L.				
mm	mm	mm	mm	mm	mm	mm	mm	mm	mm				
46	68	4	4	15 $\frac{1}{4}$	14 $\frac{1}{4}$	16 $\frac{1}{2}$	16 $\frac{1}{2}$	1	1	14 $\frac{1}{4}$	16 $\frac{1}{2}$		
47	36	5	5	18	18	16 $\frac{1}{2}$	18	1	1	19 $\frac{1}{2}$	18		
48	32	4 $\frac{1}{2}$	4 $\frac{1}{2}$	15 $\frac{1}{4}$	16 $\frac{1}{2}$	16 $\frac{1}{2}$	16 $\frac{1}{2}$	2	2	18	16 $\frac{1}{2}$		
49	36	4	4	13 $\frac{1}{4}$	13 $\frac{1}{4}$	12 $\frac{1}{4}$	14 $\frac{1}{4}$	1 $\frac{1}{2}$	1 $\frac{1}{2}$	12 $\frac{1}{4}$	13 $\frac{1}{4}$		
50	33	4	4	15 $\frac{1}{4}$	15 $\frac{1}{4}$	15 $\frac{1}{4}$	15 $\frac{1}{4}$	2	2	15 $\frac{1}{4}$	14 $\frac{1}{4}$		
51	33	4 $\frac{3}{4}$	4 $\frac{3}{4}$	10 $\frac{3}{4}$	10 $\frac{3}{4}$	12 $\frac{1}{4}$	11 $\frac{1}{4}$	1 $\frac{1}{4}$	1 $\frac{1}{2}$	11 $\frac{1}{4}$	12 $\frac{1}{4}$		
52	74	4	4	18	18	18	16 $\frac{1}{2}$	1	1	16 $\frac{1}{2}$	18		
53	77	5	5	18	18	18	18	1 $\frac{1}{2}$	1 $\frac{1}{2}$	16 $\frac{1}{2}$	16 $\frac{1}{2}$		
54	51	5 $\frac{1}{2}$	5 $\frac{1}{2}$	21	21	23	21	1	1	23	23		
55	17	4	4	19 $\frac{1}{2}$	18	19 $\frac{1}{2}$	18	1	1	18	18		
56	35	4 $\frac{1}{2}$	4 $\frac{1}{2}$	16 $\frac{1}{2}$	16 $\frac{1}{2}$	16 $\frac{1}{2}$	18	1 $\frac{3}{4}$	1	15 $\frac{1}{4}$	16 $\frac{1}{2}$		
57	35	4	4	16 $\frac{1}{2}$	14 $\frac{1}{4}$	15 $\frac{1}{4}$	15 $\frac{1}{4}$	2	2	13 $\frac{1}{4}$	12 $\frac{1}{4}$		
58	27	4 $\frac{1}{2}$	4 $\frac{1}{2}$	14 $\frac{1}{4}$	14 $\frac{1}{4}$	13 $\frac{1}{4}$	14 $\frac{1}{4}$	1 $\frac{1}{2}$	1 $\frac{1}{2}$	12 $\frac{1}{4}$	13 $\frac{1}{4}$		
59	32	3	3	13 $\frac{1}{4}$	12 $\frac{1}{4}$	14 $\frac{1}{4}$	14 $\frac{1}{4}$	1 $\frac{1}{2}$	1 $\frac{1}{2}$	12 $\frac{1}{4}$	11 $\frac{1}{4}$		
60	21	3 $\frac{1}{2}$	3 $\frac{1}{2}$	19 $\frac{1}{2}$	19 $\frac{1}{2}$	19 $\frac{1}{2}$	19 $\frac{1}{2}$	1 $\frac{1}{4}$	2 $\frac{1}{4}$	19 $\frac{1}{2}$	21		
61	40	4	4 $\frac{1}{2}$	14 $\frac{1}{4}$	14 $\frac{1}{4}$	13 $\frac{1}{4}$	13 $\frac{1}{4}$	1	1 $\frac{1}{4}$	14 $\frac{1}{4}$	11 $\frac{1}{4}$		
62	25	3	3	16 $\frac{1}{2}$	16 $\frac{1}{2}$	16 $\frac{1}{2}$	15 $\frac{1}{4}$	1	1	18	16 $\frac{1}{2}$		
63	22	4	4	18	18	18	18	$\frac{3}{4}$	$\frac{3}{4}$	18	16 $\frac{1}{2}$		
64	29	5	5	16 $\frac{1}{2}$	16 $\frac{1}{2}$	15 $\frac{1}{4}$	16 $\frac{1}{2}$	1 $\frac{1}{4}$	1 $\frac{1}{4}$	15 $\frac{1}{4}$	18		
65	22	3 $\frac{1}{2}$	3 $\frac{1}{2}$	13 $\frac{1}{4}$	13 $\frac{1}{4}$	11 $\frac{1}{4}$	13 $\frac{1}{4}$	1 $\frac{1}{2}$	1 $\frac{1}{2}$	13 $\frac{1}{4}$	13 $\frac{1}{4}$		
66	14	4	4	18	18	18	21	2	2	18	18		
67	28	4	4	15 $\frac{1}{4}$	16 $\frac{1}{2}$	14 $\frac{1}{4}$	15 $\frac{1}{4}$	1	1	15 $\frac{1}{4}$	16 $\frac{1}{2}$		
68	40	4	4	13 $\frac{1}{4}$	14 $\frac{1}{4}$	14 $\frac{1}{4}$	14 $\frac{1}{2}$	1 $\frac{1}{4}$	1 $\frac{1}{4}$	14 $\frac{1}{4}$	15 $\frac{1}{4}$		
69	41	3	3	19 $\frac{1}{2}$	19 $\frac{1}{2}$	18	19 $\frac{1}{2}$	1 $\frac{1}{2}$	1 $\frac{1}{2}$	19 $\frac{1}{2}$	21		
70	29	4	5	14 $\frac{1}{4}$	14 $\frac{1}{4}$	14 $\frac{1}{4}$	14 $\frac{1}{4}$	1	1	14 $\frac{1}{4}$	13 $\frac{1}{4}$		
71	22	5	5	23	25	21	25	1 $\frac{1}{2}$	1 $\frac{1}{2}$	21	25		
72	21	5	5	14 $\frac{1}{4}$	14 $\frac{1}{2}$	14 $\frac{1}{4}$	14 $\frac{1}{4}$	1 $\frac{1}{2}$	1 $\frac{1}{2}$	14 $\frac{1}{4}$	13 $\frac{1}{4}$		
73	42	4 $\frac{1}{2}$	5	14 $\frac{1}{4}$	13 $\frac{1}{4}$	14 $\frac{1}{4}$	14 $\frac{1}{4}$	1	1 $\frac{1}{4}$	14 $\frac{1}{4}$	13 $\frac{1}{4}$		
74	37	5	5	9 $\frac{1}{4}$	9 $\frac{1}{4}$	10	9 $\frac{1}{4}$	1	1	9 $\frac{1}{4}$	10		
75	39	4	4	12 $\frac{1}{4}$	12 $\frac{1}{4}$	12 $\frac{1}{4}$	13 $\frac{1}{4}$	1	1	14 $\frac{1}{4}$	14 $\frac{1}{4}$		
76	19	6 $\frac{1}{2}$	6 $\frac{1}{2}$	15 $\frac{1}{4}$	15 $\frac{1}{4}$	15 $\frac{1}{4}$	15 $\frac{1}{4}$	1 $\frac{3}{4}$	1 $\frac{3}{4}$	15 $\frac{1}{4}$	15 $\frac{1}{4}$		
77	37	4	3	11 $\frac{1}{2}$	11 $\frac{1}{2}$	12 $\frac{1}{4}$	12 $\frac{1}{4}$	1	1	13 $\frac{1}{4}$	12 $\frac{1}{4}$		
78	57	4 $\frac{1}{2}$	4 $\frac{1}{2}$	15 $\frac{1}{4}$	14 $\frac{1}{4}$	15 $\frac{1}{4}$	15 $\frac{1}{4}$	1 $\frac{1}{2}$	1 $\frac{1}{2}$	15 $\frac{1}{4}$	14 $\frac{1}{4}$		
79	25	3	3	15 $\frac{1}{4}$	18	18	18	1 $\frac{1}{4}$	1 $\frac{1}{4}$	16 $\frac{1}{2}$	18		
80	35	3 $\frac{1}{2}$	3 $\frac{1}{2}$	13 $\frac{1}{4}$	13 $\frac{1}{4}$	14 $\frac{1}{4}$	14 $\frac{1}{4}$	1 $\frac{1}{4}$	1 $\frac{1}{4}$	15 $\frac{1}{4}$	14 $\frac{1}{4}$		
81	36	5 $\frac{1}{2}$	5 $\frac{1}{2}$	15 $\frac{1}{4}$	15 $\frac{1}{4}$	14 $\frac{1}{4}$	15 $\frac{1}{4}$	1	1	15 $\frac{1}{4}$	15 $\frac{1}{4}$		
82	32	4 $\frac{1}{2}$	4 $\frac{1}{2}$	14 $\frac{1}{4}$	14 $\frac{1}{4}$	14 $\frac{1}{4}$	14 $\frac{1}{4}$	1 $\frac{1}{2}$	1 $\frac{1}{2}$	14 $\frac{1}{4}$	14 $\frac{1}{4}$		
83	40	5	4 $\frac{1}{2}$	12 $\frac{1}{4}$	13 $\frac{1}{4}$	12 $\frac{1}{4}$	12 $\frac{1}{4}$	1 $\frac{1}{4}$	1 $\frac{1}{4}$	11 $\frac{1}{4}$	12 $\frac{1}{4}$		
84	41	5	5	16 $\frac{1}{2}$	16 $\frac{1}{2}$	15 $\frac{1}{4}$	14 $\frac{1}{4}$	1 $\frac{1}{4}$	1 $\frac{1}{4}$	15 $\frac{1}{4}$	15 $\frac{1}{4}$		
85	22	5	5	8 $\frac{1}{2}$	9 $\frac{1}{2}$	8 $\frac{1}{2}$	9 $\frac{1}{2}$	1	1	8 $\frac{1}{2}$	10		
86	28	3 $\frac{1}{2}$	3 $\frac{1}{2}$	18	18	18	18	1 $\frac{1}{2}$	1 $\frac{1}{2}$	18	18		
87	47	4 $\frac{1}{2}$	4 $\frac{1}{2}$	19 $\frac{1}{2}$	19 $\frac{1}{2}$	18	19 $\frac{1}{2}$	1	1	19 $\frac{1}{2}$	19 $\frac{1}{2}$		
88	19	3	3	14 $\frac{1}{4}$	15 $\frac{1}{4}$	15 $\frac{1}{4}$	14 $\frac{1}{4}$	1 $\frac{1}{2}$	1 $\frac{1}{2}$	14 $\frac{1}{4}$	14 $\frac{1}{4}$		
89	27	4	4	16 $\frac{1}{2}$	16 $\frac{1}{2}$	16 $\frac{1}{2}$	16 $\frac{1}{2}$	1 $\frac{1}{4}$	1 $\frac{1}{4}$	16 $\frac{1}{2}$	16 $\frac{1}{2}$		
90	23	4	4	14 $\frac{1}{4}$	15 $\frac{1}{4}$	15 $\frac{1}{4}$	15 $\frac{1}{4}$	2	2	14 $\frac{1}{4}$	15 $\frac{1}{4}$		
91	39	4	4	18	18	18	18	1 $\frac{1}{2}$	1 $\frac{1}{2}$	18	18		
92	55	3	3	19 $\frac{1}{2}$	21	19 $\frac{1}{2}$	21	$\frac{3}{4}$	$\frac{3}{4}$	18	21		

*Intraocular Tension Not Lowered by Narrowing Pupil.* 181

CASE No.	AGE.	BEFORE MIOSIS.						AFTER MIOSIS.			
		PUPILS.		TENSION.				PUPILS.		TENSION.	
				<i>First.</i>		$\frac{1}{2}$ hour later.					
		R.	L.	R.	L.	R.	L.	R.	L.	R.	L.
		mm	mm	um	mm	mm	mm	mm	mm	mm	mm
93	47	5	5	18	18	16½	16½	I	I	16½	16½
94	50	4½	4½	15¼	15¼	15¼	15¼	I	I	14¼	13¼
95	33	3	3	18	18	19½	18	¾	¾	16½	16½
96	28	4	4	14¼	13¼	14¼	14¼	I	I	13¼	12¼
97	31	4½	4½	16½	16½	16½	16½	I	I	16½	16½
98	61	5	5	16½	14¼	16½	16¼	1¼	1¼	14¼	13¼
99	41	4½	4½	16½	16½	18	18	1½	1½	16½	16½
100	45	3½	3½	15¼	14¼	16½	16½	1½	1½	15¼	16½

## DENTAL INFECTION IN EYE DISEASES.

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WITHIN the last few years much has been written concerning the relation between dental infection and the condition generally described as "rheumatism." It has been definitely proven that various forms of arthritis are cured or materially benefited by the extraction of infected teeth or by the cleaning up of a pyorrheal condition of the mouth. The arthritis occurs, in all probability, as the result of the lodging in the joint of bacteria or of their products which have entered the blood stream from the mouth. This would explain the migratory phase of "rheumatism," distant joints being affected in rather rapid succession, and would disprove the old theory of rheumatism being due to a non-assimilation of the acid products of proteid metabolism.

As would naturally be supposed, these organisms also lodge in the eye, giving rise to infection there. In fact this is the case more frequently than has heretofore been believed. It has been estimated that 50% of all adults suffer from pyorrhea in some form and, in reporting a series of 215 eye infections due to sepsis, Lang (1) found that 139, or nearly 65%, were due to this cause. Any part of the eye may be attacked as the result of dental infection, although by far the greatest number of cases show the iris, ciliary body, or choroid to be affected. Another condition of the teeth which may give rise to infection of the eye is an apical or so-called "blind" abscess. These abscesses are quite common, Ulrich (2) having found that apical abscesses were present in 83% of dead teeth and in over 68% of artificially devitalized teeth, and their

presence is often unsuspected or undetected until the X-rays are employed. Brown and Irons (3) in their excellent paper on "The Etiology of Iritis," found dental infection to be the source of iritis in 18 of the 100 cases studied, and 41 of the cases showed apical infection or alveolar abscesses on X-ray examination, which findings were subsequently confirmed by the oral surgeon. In their study peridental infection was not included. This would exclude pyorrhea alveolaris, which according to Finnoff (4) is the most frequent cause of ocular lesions, ranking higher than blind abscesses, septic roots, or periapical abscesses. Opposed to this view is Lawler (5) who maintains that pyorrhea itself is a very infrequent cause of ocular infection. He bases his opinion on the facts that careful treatment of the mouth has failed to exert any favorable action upon the ocular condition, especially in cases of scleritis and sclero-keratitis, and that vaccine treatment has been generally disappointing.

The exciting organism of ocular infections due to oral sepsis may be any one of the variety or a combination of those found in the mouth or on the tooth roots. It is obviously impossible in most cases to determine the exact organism causing the infection in the eye as an eye is seldom enucleated as the result of such an infection, but from cultures obtained from the roots of teeth, it is fair to assume that streptococci, especially the streptococcus viridans, staphylococci, and pneumococci are the bacteria most frequently involved. Hartzell (6) has shown that the streptococcus viridans is present in chronic dental abscesses in pure culture and also in pyorrhea. Hæmolytic streptococcus is absent. These organisms have never been found in healthy tissue. In studying a series of forty dental abscesses, Gilmore and Moody (7) found that streptococci were the organisms most constantly present and Hartzell and Henrici (8) maintain that streptococci of the viridans group are always present in peridental suppurative lesions. They are normally of low virulence but are at times able to produce lesions of the heart, aorta, kidneys, and joints. They are constantly present in lesions presenting a large ulcerated surface through which they may pass into the deeper tissues and the blood stream and cause metastases. The exact identity of the streptococcus viridans is still in

doubt. Hartzell of the University of Minnesota considers the streptococcus viridans of Schottmueller to be the same organism as the streptococcus brevis of Van Lingelsheim, the streptococcus salivarius of Andrewes and Horden, and to be identical with the diplococcus rheumaticus of Poynton and Paine. Edward C. Rosenow of the Mayo Foundation is a radical opponent of this theory. He asserts that the streptococcus viridans can change into the streptococcus pyogenes on the one hand and the pneumococcus on the other. The pus-producing cocci are not permanent inhabitants of the mouth. Their presence is considered accidental. Moody found the staphylococcus aureus eighty times in one thousand mouths, but found the staphylococcus pyogenes albus which possesses low virulency frequently in suppurating conditions of the mouth. The so-called "blind" abscesses at the apex of a tooth may become purulent by the staphylococci gaining entrance to that region. Henrici has attempted to demonstrate the pathogenicity of dental streptococci by complement fixation test, as has been done by Besredka, Hastings, and others. He concludes that the results have been negative because the patients do not produce antibodies in sufficient quantity to be demonstrated by the method of complement fixation and that unless the local foci are removed a vaccine will hardly be of benefit.

A most interesting phase of infection is the selective action possessed by bacteria and this has been very carefully studied by Rosenow. He found that streptococci obtained from the appendix if injected into animals most often produced lesions in that organ. Also, that streptococci isolated from arthritis more often affected the eye than those isolated in gastric and duodenal ulcers and that organisms from the joint show a selective action for the eye. The selective tissues of mouth organisms have been the joints, endocardium, and eye. Irons (9) in studying a series of 329 unselected cases variously grouped as to disease, X-rayed 124. In 44% of these 124 alveolar abscesses were found. In the arthritic group alveolar abscesses were present in 76%; in the nephritis-cardio-vascular group they were present in 47%; and in the group comprising other diseases, such as pneumonia, respiratory and gastro-intestinal, 23%. Abnormalities in tonsils were present



in 45% of the arthritic group, in 24% of the nephritis-cardio-vascular group, and in 19% of the remainder. Other chronic conditions such as sinus infections or infections of the genito-urinary tract were present in 21% of the arthritic group, 13% of the nephritis-cardio-vascular group, and in 11% of the other diseases. Syphilis was found in 23% of the arthritic group, 39% of the nephritis-cardio-vascular group, and 13% of other diseases. He considers the use of vaccines doubtful, at best being only an adjunct to other forms of treatment. From these figures it would seem that while there may not be a direct relationship between alveolar abscesses and the various diseases enumerated, the percentage of coincidence is far greater.

The exact mode of eye infection from a dental focus is still a debated question. Numerous theories have been advanced. Lelongt holds that infection takes place through channels in the bone, subperiosteal tissue, or lymphatics. Polet thinks that in addition to extension through the subperiosteal and osseous channels infection is transmitted along the nerves. Dutoit believes that infection can be carried from the dental alveolus through the alveolar plexus and the pterygoid plexus into the ophthalmic vein and cavernous sinus. Ulrich (2) and Dimmer (10) believe that the infection is always hematogenous and Finnoff regards this as the only possible mode of deep-seated infection as the lymphatics of the mouth drain into the mandibular and cervical glands. A direct extension through the venous channels from the diseased area to the cavernous sinus is anatomically possible and unquestionably has produced a sinus thrombosis and its symptoms.

There is no definite clinical picture that can be attributed to dental infection. Any part of the eye may be affected, but the iris, ciliary body, and choroid are most frequently involved. Ibershoff (11) states that "the cases can be classed as sub-acute and chronic serous cyclitis with the formation of exudates on the posterior layer of the cornea, in the aqueous and vitreous. The cases are long-standing, mild in character, devoid of pain or other manifestations associated with acute inflammatory processes, and have involved the iris but little or not at all." This represents a true picture of dental infection with the possible exception of frequency of iris involvement. The iris is probably more frequently affected than

would be supposed from his statement. While a purulent exudate is at times present, it is exceptional. In a diffuse suppurative pyorrhea with an abundance of pus in the mouth it is only logical to assume that the accompanying eye infection would also be of a purulent type. The reason why this is rarely so is explained by de Schweinitz (12) who states that "although the primary lesion is suppurative it produces a non-septic focus because the organisms decrease in virulence in their contact with the blood stream and they are not contained in emboli or blood clot."

Since the establishment of a dental department at the Herman Knapp Memorial Eye Hospital two years ago the investigation of dental infection has received particular attention and the following report represents the work of the first year and a half. The work was carried on in collaboration with Dr. Joseph M. Levy, who performed the dental work, and Dr. Marshall Carleton Pease, Jr., who performed the bacteriological work. In all fifty-three cases were examined, the diagnosis of these cases being as follows: acute iritis and iridocyclitis, 13; acute and chronic choroiditis, 10; chronic iridocyclitis, 9; post-operative iritis, 4; detachment of retina, 4; episcleritis, 2; dendritic keratitis, 2; and one case each of chorioretinitis, chronic cyclitis, irido-choroiditis, interstitial keratitis (non-specific), vesicular keratitis, kerato-iritis, retrobulbar neuritis, retinal hemorrhage, and ulcer of the cornea. The routine treatment of cases where other infections are excluded is to have the teeth X-rayed, the blood examined, the streptococcus complement fixation test being employed, the teeth treated and, if necessary, extracted, and a vaccine prepared from the culture obtained at the time of extraction.

In examining this series of cases fifty-two of the fifty-three patients showed dental infection on the same side as the affected eye. This would lead to the belief that infection does not occur through the blood stream, as if this were so it would seem that more cases in which dental infection was present on the opposite side of the affected eye would have been noted. In view of this observation it is a logical conclusion that infection is carried either by means of the lymphatics or through osseous channels.

It was found that of the 53 cases 15 were mixed infections. One patient, having a + + + + Wassermann reaction in addition to infected teeth, was treated with mercury with no improvement. As soon as dental treatment was instituted the condition cleared up rapidly. Two cases showed a positive Wassermann reaction and a reaction to tuberculin besides having infected teeth. In the one having a + + + + Wassermann no improvement took place as the result of specific or dental treatment. In the other having a + + Wassermann reaction no mercury was given, but improvement took place as the result of dental treatment. The remaining 12 cases reacted positively to tuberculin in addition to having infected teeth. Of these, 6 received both tuberculin injections and dental treatment, 3 cases showing no improvement at all, 2 showing improvement as the result of dental treatment, and in 1 case it was impossible to say whether the dental treatment or the tuberculin injections caused the improvement. Of the remaining 6 cases 2 were improved as the result of dental treatment and 4 showed no improvement whatsoever.

The streptococcus complement fixation test was employed in 40 cases, being positive in 24 or 60% and negative in 16 or 40%. Of the 24 giving a positive reaction, 4 or 16 + % were cured; 14 or 58 + % were improved, and in 6 or 25% there was no improvement. Of the 16 cases giving a negative reaction, 3 or 18 + % were cured; 8 or 50% were improved, and in 5 or 31 + % there was no improvement. Of the 4 cured cases giving a positive reaction 2 showed streptococcus viridans in the cultures and received vaccines, no cultures being made in the remaining 2. Of the 14 improved cases giving a positive reaction 4 showed streptococcus viridans in culture, 1 of whom received vaccines; 2 showed staphylococcus in culture and received vaccines; 1, a case of severe pyorrhea, received stock vaccines, and in the remaining 7 cases no cultures were made. Of the 6 cases giving a positive reaction and showing no improvement, 2 showed streptococcus viridans in culture and received vaccines, no cultures being made in the remaining 4 cases. Of the 3 cured cases giving a negative reaction, 1 showed streptococcus viridans in culture but received no vaccines; in the other 2 cases no cultures were

made. Of the 8 cases showing improvement and giving a negative reaction, 4 showed streptococcus viridans in culture, 2 of whom received vaccines, and in the remaining 4 cases no cultures were made. Of the 5 cases giving a negative reaction and showing no improvement, 3 showed streptococcus viridans in culture and received vaccines, and in the remaining 2 no cultures were made.

In 25 cases bacteriological cultures were made either from the roots of the teeth extracted or from the tooth sockets. 14 or 56% showed a pure culture of streptococcus viridans; 2 or 8% showed a pure culture of staphylococcus; 4 or 16% showed a mixture of streptococcus and staphylococcus; 3 or 12% showed a mixture of streptococcus, staphylococcus, and bacillus pyocyaneus; 1 or 4% showed a mixture of streptococcus, staphylococcus, and pneumococcus, and 1 or 4% showed a mixture of streptococcus, staphylococcus, bacillus pyocyaneus, and pneumococcus. In analyzing the 14 cases in which a pure culture of streptococcus viridans was obtained it was found that 10 were of a chronic nature, the remaining 4 being made up of 3 cases of post-operative iritis and 1 case of acute iritis. This would seem to bear out Hartzell's theory that the streptococcus viridans is an organism of diminished virulence. Of the 2 cases which showed a pure culture of staphylococcus, 1 was an acute iridocyclitis, the other an exudative choroiditis. Of the 4 cases showing both streptococcus and staphylococcus, there was one each of detachment of the retina, acute iritis, post-operative iritis, and retinal hemorrhage. Of the 3 cases showing streptococcus viridans, staphylococcus and bacillus pyocyaneus, there were 2 cases of exudative choroiditis and one of chronic iridocyclitis. The case showing streptococcus, staphylococcus, and pneumococcus was one of episcleritis, and the case showing streptococcus, staphylococcus, bacillus pyocyaneus and pneumococcus was one of exudative choroiditis.

Vaccines were administered to 18 patients, 17 receiving autogenous vaccines and one stock vaccines. 2 or 11 + % of these cases were cured; 10 or 55 + % were improved, and in 6 or 33 + % there was no improvement. Both the cured cases gave a positive streptococcus complement fixation test, 1, a case of chronic iridocyclitis, showing a pure culture of

streptococcus viridans: the other, a case of episcleritis, showing streptococcus, staphylococcus: and pneumococcus in the culture. Of the 10 cases showing improvement, 2 were cases of post-operative iritis, showing a positive complement fixation test and a pure culture of streptococcus viridans; 2, cases of chronic iridocyclitis, 1, in which the complement fixation test was not made, showing streptococcus, staphylococcus, and bacillus pyocyaneus in culture, the other, in which the complement fixation test was negative, showing streptococcus viridans in culture. The other cases were: acute recurrent iritis, in which the complement fixation test was not made, but which showed streptococcus viridans and staphylococcus in culture; vesicular keratitis, with a negative complement fixation test, and showing streptococcus viridans in culture; a case of acute iridocyclitis with a positive complement fixation test, showing staphylococcus in culture; a case of corneal ulcer with a positive complement fixation test and which received stock vaccines (staphylo-acne); a case of chorioretinitis, in which the complement fixation test was not made, showing streptococcus viridans in culture, and a case of choroiditis with a positive complement fixation test, showing staphylococcus in culture. Of the 6 cases in which no improvement was obtained as the result of vaccine treatment, there were 2 of detachment of the retina, both giving a positive complement fixation test and showing streptococcus viridans in culture; 2 of chronic iridocyclitis, 1 having a positive and 1 a negative complement fixation test, but both showing streptococcus viridans in culture; 1 of choroiditis with a negative complement fixation test and showing streptococcus, staphylococcus, bacillus pyocyaneus, and pneumococcus in culture, and 1 of retinal hemorrhage, in which the complement fixation test was not made, and showing streptococcus and staphylococcus in culture.

Besides the elimination of the dental focus the patients received the usual treatment indicated in the various conditions, such as, atropine, leeching, sodium salicylate, etc. Of the 53 cases, 7 or 14 + % were cured; 27 or 50 + % were improved, and in 19 or 35 + % there was no improvement. 24 of the 53 cases were of an acute nature and 29 were of a chronic nature. As might be expected, the cases which

showed the most benefit as the result of dental treatment were acute conditions, while the cases in which no improvement was noted were mostly of a chronic nature. Of the 24 acute cases 4 or 16 + % were cured; 18 or 75% were improved, and in 2 or 8 + % there was no improvement. Of the 29 cases of a chronic nature, 3 or 10 + % were cured; 9 or 30 + % were improved, and in 17 or 58 + % there was no improvement.

In summarizing the results of the investigation the following conclusions are reached:

(1) Dental infection does not present a definite clinical entity, although it is most often seen in the form of a low-grade chronic infection involving the iris, choroid, and ciliary body.

(2) Dental infection is more frequent than has heretofore been believed and it must be carefully sought for by means of the X-ray.

(3) The infection is carried to the eye in all probability through the lymphatics and osseous channels.

(4) The streptococcus viridans is the organism most frequently found in cultures from tooth roots or sockets and is the main bacteriological factor. Other organisms, as the staphylococcus, pneumococcus, and bacillus pyocyaneus, must also be considered.

(5) In acute cases a mixed infection is the rule, the streptococcus viridans being most often associated with the staphylococcus; whereas in chronic cases the viridans alone is common.

(6) The streptococcus complement fixation test has not given satisfactory results, the findings not being consistent with the clinical picture.

(7) Vaccine treatment has been disappointing. The percentage of cures in cases receiving vaccines was only 11 + % as compared to a general percentage of cures of 14 + % of the cases treated. The administration of vaccines in acute cases gave better results than in chronic cases and those cases presenting a positive streptococcus complement fixation test with streptococcus viridans in the culture seemed to be benefited more by the administration of vaccines than others.

(8) The factors most concerned in the cure or improvement of these cases were the extraction of the offending teeth and the resulting drainage of the infected sockets.

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## EYE MANIFESTATIONS IN A CASE OF POLYCYTHÆMIA.

By DR. MARTIN COHEN, NEW YORK.

**M**RS. E. J., 60 years of age, family history irrelevant. In the past history, one spontaneous miscarriage, and deafness in the left ear for the past three years are the only noteworthy facts.

The condition reported dates back two years, when she first came under observation at the Post-Graduate Hospital. At that time she complained of failing vision in the right eye, hot flushes, and other symptoms of polycythæmia, and also manifestations of an arteriosclerotic involvement of the kidney.

Physical examination showed plethora and cyanosis of the skin and mucous membrane, a moderate degree of emphysema, a normal cardiac condition, and an enlarged spleen and liver. There were no other glandular changes.

Blood pressure, slightly increased; the systolic, 175; diastolic, 85.

The urine was scanty, 300 to 900cc, and of low specific gravity; there were a few granular casts and a trace of albumin, characteristic of a chronic interstitial nephritis.

The phenosulphothalein test was about normal (40 per cent.). The blood contained between 8 and 10 million red blood cells, which is double the normal quantity, and a hæmoglobin of 140 to 160 per cent. The white blood cells were relatively normal, except for a slight increase of lymphocytes; there were no abnormalities of the other elements or blood platelets; the coagulation and bleeding time were normal. The viscosity test was not determined. The chemical examination indicated no acidosis.

A finding of considerable importance was a strongly positive Wassermann blood test. The treatment consisted mainly of venesection of 900cc of blood, followed by an infusion of 500cc of saline solution; this treatment was



repeated after a few weeks and resulted in a lessening of the degree of cyanosis and an improvement in the general symptoms, including the venous hyperæmia of the retinal vessels.

The examination of the fundi two years ago showed a thrombosis of the right central retinal vein, probably not due to the mild nephritis or the mild arteriosclerosis, and a fundus polycythæmicus of both eyes. At present, the ocular examination is as follows:

Right eye, no light perception; pupil slightly dilated, rendering no response to direct examination but giving an indirect light reaction; no abnormal deviation of the eyeball; the intraocular tension is normal; the tarsal conjunctivæ and caruncles are hyperæmic; the papilla is of a steel gray color, its nasal border alone is moderately blurred; the macular region is studded with a few ill-defined grayish and pigmented spots.

The vascular system is practically similar in both eyes. The retinal veins are characteristic of the polycythæmic condition; they are markedly dilated, their distension extending far into the periphery of the fundus; they are also uneven in caliber, have a distinctly bluish appearance, and several U-shaped indentations of the entire vessel wall occur along their course,—which are independent of the arterial compression of the veins; along one of their borders and at some distance from the disk, the superior and inferior retinal veins are surrounded by whitish streaks which are probably due to a lymphstasis. Venous compression due to arterial crossing is observed over a few places of the fundus. The arteries are apparently increased in number on and near the disk, which is due to an injection of the normal and invisible branches; they are also tortuous, normal in caliber, and their central light reflex streak is prominent.

Left disk: Its color is yellowish white on the temporal side, with a sharply outlined margin, but the nasal side is elevated to three diopters. Fundus findings: fundus polycythæmicus, R. and L.; retinal arteriosclerosis R. and L.; post-neuritic optic atrophy and central chorioretinitis R., and papillitis in left eye. Although one would expect to find a retinal cyanosis in polycythæmia, still this case is of interest for the following reasons:

1. The association of lues with polycythæmia is rare; still, in this case the combination might be coincidental.
2. The presence of a strongly positive Wassermann reaction, a bilateral retinal arteriosclerosis, and a unilateral central

chorioretinitis suggests the presence of a specific retinal arteriosclerosis.

3. Thrombosis as a complication of polycythæmia has been described, but its location in the central retinal vein in this condition has not, to my knowledge, been previously recorded in the literature.

4. At a recent section meeting I reported a case which demonstrated the increase of the white blood cells in the blood with leukæmic manifestations in the retina and choroid; this case is almost a counterpart, showing increase of the red blood cells in the blood and characteristic polycythæmic manifestations in the retina.

## REPORT OF THE TRANSACTIONS OF THE OPHTHALMOLOGICAL SECTION OF THE ROYAL SOCIETY OF MEDICINE.

BY MR. H. DICKINSON, LONDON.

An ordinary meeting of the Section was held on Wednesday, December 12, 1917, Mr. WILLIAM LANG, F.R.C.S., the President of the Section, being in the Chair.

### **Disturbances of Vision by Cerebral Lesions.**

The whole sitting was devoted to the reception of an important contribution on this subject by Lieut.-Colonel GORDON HOLMES, M.D., who illustrated his thesis by means of many photographs and diagrams. It followed the contribution, eighteen months ago, of the paper by Colonel Lister and the present author, before the same body, on the disturbances of vision produced by gunshot injuries of the visual cortex and of the optic radiations. Certain conclusions on the cortical representation of the retina were evolved, especially the segmental correspondence of different areas of the retina with separate zones of the cortical visual area. The main conclusions set out in the former paper were:

1. The upper half of each retina is represented in the dorsal, the lower in the ventral part of each visual area.

2. The center for macular or central vision lies in the posterior extremities of the visual areas, probably in the margins and the lateral surfaces of the occipital poles. The macular region has not a bilateral representation.

3. The center for vision subserved by the periphery of the retina is probably situated in the anterior end of the visual

area, and the serial concentric zones of the retina from the macula to the periphery are probably represented in this order from behind forwards in the visual area.

The foregoing agree with those already arrived at by Inouye from a study of cases in the Russo-Japanese war, and by others. The author has now seen a large number of cases since the last communication to the Section, and all the observations on these harmonized with the foregoing conclusions.

Central and paracentral scotomata are very common in all moderate injuries of the occipital lobes. Injury of the poles of both hemispheres may cause complete loss of central vision; an unilateral wound produces homonymous scotomata in the opposite halves of the fields. A common type of case is that of pure lateral paracentral scotomata, of which an instance was that of a man whose helmet was penetrated by a shrapnel ball, rendering him quite blind until next day. No subjective visual phenomena were noticed. From a small penetrating wound, softened brain tissue extruded, just to the right of the mid-line, and one inch above the inion. The radiograph showed much depressed bone, but no foreign body. After the removal of brain tissue and bone from the wound, the sight was quickly recovered. A week later, there was a large left homonymous paracentral scotoma, but peripheral vision was unaffected and the color fields were normal in the right halves, but there was no perception of either red or green to the left of the middle line.

In all patients with inferior unilateral paracentral scotomata the injury involved the tip or posterior portion of one occipital lobe, at or immediately above the level of the calcarine fissure, and usually the size of the scotoma had a relation to the depth of the wound. Some cases of bilateral inferior scotomata afford valuable evidence on the subject of cortical localization. Isolated superior paracentral scotomata are much less common, and in the few cases seen by various people the lesion appeared to involve the lower part of the area striata: indicating, therefore, that the retina immediately below the macula is represented in the lower portion of the calcarine area. There seems to be sufficient evidence to allow the conclusion to be formed that the upper halves of the retina are projected on to the

dorsal part of the visual area, and the lower halves on to its ventral portion. Macular and perimacular vision are represented in the most posterior part of this area. Still, other problems require consideration, and they can best be studied in cases with paracentral scotomata. Usually, the sector scotomata lie between the vertical line through the fixation-point and one of the oblique radii.

The author then proceeded to deal with the cortical representation of peripheral vision. In the previous communication, Colonel Lister and he could only suggest, by a process of exclusion, that vision in the periphery of the fields was represented in the anterior portion of the cortical visual area. It was not to be expected that many cases suitable for giving definite evidence on this would be seen, as perforating wounds which could involve the anterior portion of the area striata would very likely involve the optic radiations also, and so cause intensive or irregular areas of blindness. A recent case which came under Riddock's observation, however, gives valuable positive evidence of the localization of peripheral vision. This man had considerable peripheral contraction of both visual fields. A rifle bullet and, later, fragments of bone were removed from the great longitudinal fissure between the two hemispheres, at such a level and depth that the anterior portion of the calcarine area was probably injured. The author himself had recently seen a case which was valuable from the point of view of the elucidation of this question, and he narrated it in detail. The cases seen afforded some positive evidence that peripheral vision is represented in the anterior portions of the calcarine area. When these observations are associated with the fact that peripheral vision is never affected by local lesions of the posterior part of this area, the hypothesis becomes very probable if it be admitted that the visual sphere at least is roughly approximate with the area striata.

Destructive injuries of the optic radiations generally produce a complete hemianopia, in which the blindness may or may not reach the fixation point, or large irregular areas of blindness. Occasionally the visual defect is more regular, and these affect the important question whether the fibers of the radiations which carry impressions from the retina and

end in definite portions of the visual cortex, are arranged in order according to the origin of the impressions they carry and to their exact termination. The author has seen a number of cases in which quadrantic hemianopias or other defects of the fields resulted from wounds of the radiations; some of these he quoted. The author was forced to the conclusion that the fibers of the radiations are arranged regularly in laminae or series, according to the impulses they carry. With regard to the course in the radiations of the fibers concerned with macular vision, observations suggest that these have a distinct path of their own, but he had not yet seen a case in which a paracentral scotoma, or an isolated affection of central or pericentral vision could be attributed to a lesion of the radiations.

Colonel Holmes then discussed the nature of the lesions. With regard to concussion, in many of his patients the defects remained unaltered at repeated examinations, though these examinations were extended over considerable periods. In others, however, there was a progressive diminution of the blind area, sometimes rapid, at other times slow. In such a highly-organized function as that of sight, recovery can hardly ever be due to vicarious representation or to the assumption of the function of the destroyed cortical areas by other centers. When the defects persist for a considerable time after the infliction of the wound, he did not doubt they depend on a simple destructive lesion. But in the earlier stages of gunshot injuries of the head it is not uncommon to find a complete hemianopia or a large area of total blindness disappear in a few days or weeks, probably by recovery from the center to the periphery. But peripheral vision often reappears before the central or pericentral. He attributed the condition when recovery was quick to an cedematous swelling of the areas concerned, analogous to that which occurs in the myelin sheaths of the spinal cord. He could not say whether a molecular disturbance might contribute to the defect. The rapidity of recovery argued a condition short of the destruction of anatomical integrity. It had been common to find a zone of partial vision around the scotomata, or on the borders of a quadrantic or incomplete hemianopia. But in most of the cases examined more than three weeks after the onset of the

blindness the margins of the scotomata were remarkably sharp. He passed on to discuss the mechanism and function of peripheral vision, and thence considered the disturbances of color perception caused by cerebral lesions. He said it had not been conclusively shown that color perception may be completely lost in any part of the field when that of light or white is undisturbed; many of the hemiachromatopsias have been instances of hemiamblyopia. He had frequently observed among his cases that red and green test-objects could not be recognized in certain regions, often in homonymous halves of the visual fields, but in every instance visual sensibility to white test-objects of the same size was reduced. His observations led him to the conclusion that an isolated loss or dissociation of color vision is not produced by cerebral lesions.

The paper concluded with a discussion of disturbances of visual attention, and loss of visual orientation and appreciation of depth. He summarized the conclusions arrived at—which, he said, could at least serve as a working hypothesis for further investigations—in the following way:

1. The upper half of each retina is represented in the dorsal, and the lower half in the ventral part of each visual area.

2. The center for macular or central vision lies in the most posterior part of the visual areas, probably on the margins and in the lateral surfaces of the occipital poles. The macula has not a bilateral representation.

3. The center for vision subserved by the periphery of the retinae is situated in the anterior portions of the visual areas, and the serial concentric zones of the retinae from the macula to the periphery are probably represented in this order from behind forwards in the visual areas.

4. Those portions of the retina adjoining their vertical axes are probably represented in dorsal and ventral margins of the visual areas, while that in the neighborhood of the horizontal axes is projected on to the walls and the floor of the calcarine fissures.

5. Severe lesions of the visual cortex produce complete blindness in the corresponding portions of the visual fields, or, if incomplete, an amblyopia, color vision being generally

lost, and white objects appearing indistinct; or only more potent stimuli, such as objects moving sharply, may excite sensations.

6. The defects of vision in the fields of the two eyes are always congruous and superimposable, provided that no disease or injury of the peripheral visual apparatus exists.

7. Lesions of the lateral surfaces of the hemispheres, particularly of the posterior parietal regions, may cause certain disturbances of the higher visual perceptual functions with intact visual sensibility, as loss of visual orientation and localization in space, disturbance of the perception of depth and distance, visual attention loss, and visual agnosia.

On the motion of Dr. JAMES TAYLOR, past-president of the Section of Neurology, the author was the recipient of a special vote of thanks for his contribution.



REPORT OF THE PROCEEDINGS OF THE SECTION  
ON OPHTHALMOLOGY OF THE NEW YORK  
ACADEMY OF MEDICINE.

BY LEWIS W. CRIGLER, M.D., SECRETARY.

OCTOBER 15, 1917. GEORGE H. BELL, M.D., CHAIRMAN.

Dr. J. BRUDER presented a patient suffering with a **gumma of left lower lid**. Man, age 34, gave a history of having contracted syphilis twelve years previously. Two months ago he noticed a swelling of left lower lid which lasted for a few days and then became ulcerated. When first seen by Dr. Bruder a month ago, he presented the following condition:

An irregular, eroded, and punched out ulcer near the outer canthus and extending to the palpebral margin. The preauricular gland was swollen. The floor of the ulcer was covered with a dirty gray exudate. The conjunctiva in the region of the wound was slightly thickened. Wassermann reaction, + + + + positive.

DISCUSSION: Dr. A. E. DAVIS called attention to the fact that malignant growths of the lid simulate, clinically, gummas, and that a thorough physical and microscopical examination of the lesion is often essential.

Dr. HELLER spoke of an accidental inoculation of the lid from a vaccination pustule on the arm of a patient which showed some of the characteristics of a gumma. Dr. Bruder said that a more general use of the Wassermann reaction, in patients presenting various syphilitic stigmata, should be performed as a prophylactic measure against future luetic involvements.

Dr. J. BRUDER presented a case of **keratitis profunda**. Patient, female, age 24, was first seen by Dr. Bruder in June,

1917. She presented the following symptoms: sudden attack of pain and obscuration of vision in left eye; severe frontal headache on left side. Her vision was reduced to hand movements, the right remaining normal. Tension normal in affected eye. Examination with loupe revealed a diffuse uniform gray opacity, situated in the center of the cornea. The opacity is seen to be composed of numerous maculae and interlacing striæ with an ill-defined border. Inflammatory symptoms were rather mild, while the irritative symptoms were quite marked. After a few days the opacity spread over the cornea, leaving only a narrow rim inside the limbus which remained clear. No deep vessels were present. Instillation of fluorescein produced a deep stain of the cornea. Wassermann and tuberculin tests were negative. X-ray examination of the teeth showed disease of left upper and second lower bicuspid, at the apices. The sinuses were normal.

DISCUSSION: Dr. REIDEL was of the opinion that thyroid-extract treatment should be tried in this case in view of the negative clinical findings.

Dr. H. H. Tyson stated that one year ago he had under treatment an eye, similar to Dr. Bruder's case, in a robust Irishman, in which all causes could be excluded except his teeth. He had a severe case of pyorrhœa alveolaris but would not consult a dentist or receive any treatment for his mouth or teeth. Notwithstanding this fact, under local ocular treatment and with Fowler's solution internally, the keratitis which lasted six months finally disappeared, leaving him with practically normal vision. The interesting point was that in spite of the continued presence of the supposed cause, the eye completely recovered.

Dr. GEO. H. BELL referred to the examination of the teeth, tonsils, and gastro-intestinal tract as a source of focal infection in these cases, when the usual physical examinations were negative, and also reported satisfactory results in a few cases when treated along these specific lines. He said that in as much as the ætiology of this rare condition was so obscure, it was important to avail one's self of all the means of diagnosis obtainable.

Dr. BRUDER presented three cases of **retinitis pigmentosa**.

Dr. GEO. H. BELL presented a case of **senile keratosis** of the

**upper lid.** Patient came to his clinic at the Infirmary with a thickened upper lid which looked like thickened epidermic patches. The elevations felt rough and horny. The patient was referred to the Skin and Cancer Hospital for diagnosis. It was there pronounced **senile keratosis, secondary to xanthoma.**

The prognosis is that it may remain quiescent or later undergo epitheliomatous degeneration. An ointment of ammoniated mercury was prescribed.

Dr. MARTIN COHEN presented a case of **polycythæmia with eye manifestations.** Mrs. E. J., age 60 years, has a mild form of chronic interstitial nephritis, arteriosclerosis, enlargement of the liver and spleen, a strongly positive Wassermann reaction, and a typical blood picture and physical signs of polycythæmia. Although one would expect to find similar changes in the eye as in the general condition in polycythæmia, still this case is of interest for the following reasons:

First, the association of polycythæmia with lues.

Second, the presence of a specific retinal arteriosclerosis.

Third, thrombosis as a complication of polycythæmia has been described, but not its location in the central retinal vein. Thrombosis of the central veins occurs in nephritis or arteriosclerosis or both, but these conditions were of a mild type in this patient, and therefore would not likely result in the fundus findings as mentioned.

**DISCUSSION:** Dr. TYSON in discussing Dr. Cohen's case took exception to his statement that the appearance of the eye backgrounds in his case was pathognomonic of polycythæmia. He considered it a case of angiosclerosis of the retinal vessels and that it was the same picture often presented in senile angiosclerosis. He did not doubt but that polycythæmia existed in the case, but did not agree with Dr. Cohen that it was the cause of the ocular condition. He said that similar cases had been reported by Oatman and others in which no polycythæmia existed.

Dr. WOORON remarked that the case represented an angio-pathic sclerosis, but that he was not acquainted with the appearance of fundi in polycythæmia.

Dr. COHEN stated that there were undoubtedly angiosclerotic changes in the retina, but that the association of polycy-

thæmia caused typical changes in the veins which were pathognomonic of polycythæmia.

Dr. H. H. TYSON reported a case of **traumatic paralysis of the left abducens nerve**, caused by injury following a fall.

History: Lieut. E. C. B., Royal Flying Corps, age 23 years, while flying at an altitude of 400 feet, on Sept. 13, 1917, was struck by another plane, both planes falling to the ground. Two officers were killed, Lieut. B. escaping with a fractured clavicle and nose and extensive orbital, subconjunctival, and subcutaneous hemorrhages, with a contused wound of left brow. On Sept. 21, 1917, vision each eye  $\frac{2}{20}$ ; had homonymous diplopia in his entire left field and extending slightly into his right field. Pain on efforts at extreme rotation to right or left. X-ray negative, fundus, tension, and vision normal. On Sept. 27th single vision existed in entire right field and ten degrees to left of median line. Oct. 10th, diplopia begins at 20 degrees to left of median line; O. S. abduction  $35^{\circ}$ —adduction,  $50^{\circ}$ ; O. D. abduction  $45^{\circ}$ , adduction  $55^{\circ}$ . Pain in left eye on attempts at extreme rotation. Pupils equal, reactions normal. No ptosis. Still moderate amount of subconjunctival hemorrhage is present four weeks after date of injury. The paralysis of the abducens in this case is evidently caused by pressure, and on account of the long course traveled by the nerve, it would be difficult to state the exact site of the injury, but considering the steady improvement in motility coincident with the disappearance of the hemorrhage it is safe to assume that it is of orbital origin and that the prognosis is good. The history of similar traumatic cases reported is that they require from six to twelve weeks for complete recovery, while there are a few which recover only partially. Sodium iodide was given internally.

DISCUSSION: Dr. LESZYNSKY considered the cause as due to a nuclear hemorrhage on account of the involvement of an isolated muscle and with no evidence of fracture.

Dr. COHEN was of the opinion that the paralysis was due to a partial severing of the nerve and not a hemorrhage in the orbit or nuclear center of the sixth nerve.

NOVEMBER 19, 1917.

Dr. BEN WITT KEY presented a case of **extensive penetrating**

**wound of the globe.** The case was that of a boy 19 years of age who had been struck in the left eye by the end of a wire hoop eleven days before presenting himself for treatment. The cornea had been penetrated from the outer limbus directly across to the nasal limbus in a horizontal line just above the pupillary margin and extending into the sclerotic at the nasal side for about 2mm. The line of perforation measured 11mm. The iris was prolapsed in the nasal third of the wound and incarcerated with the exception of about 2mm of the lower margin. The iris was freed through wound dissection, iridectomy, and replacement. A conjunctival flap was then made covering the wound and recovery was uneventful, leaving a scar which is covered by the upper lid and a moderate amount of astigmatism. Vision with correcting lenses was  $\frac{2}{30}+$  when last examined.

**DISCUSSION:** Dr. CRIGLER said that several years ago he had a case very similar to the case presented by Dr. Key: A young man, while playing handball with his myopic glasses on, received a blow which struck the right glass. The glass broke and was driven into the eye. A triangular cut was made in the cornea, one prong of which extended horizontally through the sclera several mm behind the ciliary body. There was escape of both aqueous and vitreous on removal of the piece of glass. The prolapsed iris was cut off and pillars replaced. There was no prolapse of ciliary body or choroid. The scleral wound was closed by conjunctival sutures only, with the exception of one stitch placed through the episcleral tissues at the limbus. Both eyes were bandaged. Recovery was rapid without the least signs of inflammation, and the remarkable feature in the case is, that, although both the anterior and vitreous chambers were opened the lens was not injured. Vision was restored to  $\frac{2}{30}+$  with correcting lenses.

Dr. KEY presented a case of **extensive ciliary staphyloma** in a man 62 years of age. He gave a history of having been struck in the right eye forty years ago. Since that time the eye has been blind but there have been periodic attacks of pain. Five months ago the eye became very painful and began to "swell." The appearance now is that of almost complete ciliary staphyloma. The cornea is infiltrated with calcareous deposits. Dr. Key has advised that the eye be removed.

Dr. S. T. HUBBARD presented a case of **optic neuritis of accessory sinus origin followed by complete recovery** after opening and draining these cavities. Patient, female, age 25, was seen for the first time in April, 1916. Her symptoms then were pain and tenderness of the left eye aggravated by movement, intense headache, dizziness in walking, and marked blurring of her vision. Her previous history was that of a pus appendix and a suppurating ear. Her sight grew steadily worse and at the end of a week from onset her direct vision was lost completely, with only blurred peripheral vision remaining. With this latter symptom she suffered with intense headache which was aggravated by coughing.

Examination of the backgrounds at this time revealed the right eye to be practically normal; vision  $\frac{20}{40}$ . The left eye showed a swelling of the disk with small hemorrhages present. There were also hemorrhages in the adjacent retina on the temporal side. There was a relative central scotoma. X-ray by Dr. Dixon revealed rather doubtful signs of active inflammation although there was some haziness in the frontals and sphenoids. Vision at this time was reduced to fingers at eighteen inches.

The patient was referred to Dr. Craig who opened and thoroughly drained the accessory sinuses involved. Within two weeks from date of operation the retinal œdema and hemorrhages had disappeared and the patient's vision restored to practically normal.

One year after the trouble in the left eye, the same condition developed in the right eye. It was treated in a similar manner as the left with like results. At the present time there is a contraction of the fields for color and an enlargement of the normal blind spot in both eyes.

Dr. CHARLES MACK presented a case of **hole in the retina at the macula**. The case presented no unusual features. The patient gave a history of having been struck in the right eye with a cork six years previously. There is a central scotoma of  $10^{\circ}$ .

DISCUSSION: Dr. WOOTTON stated that after the lapse of years these holes sometimes become surrounded by colloid dots.

Dr. COHEN referred to cases that had been reported of idiopathic origin in which the lesion was bilateral.

Dr. J. H. CLAIBORNE presented a case of **glaucoma success-**

fully operated on by iridectomy for the object of bringing about a discussion on the latest and best mode of procedure in the treatment.

DISCUSSION: Dr. WOLFF said that iridectomy was still the operation of choice in the treatment of acute glaucoma, but that it was better to control the initial attack by the aid of miotics if possible and then perform iridectomy at the more favorable time when the congestion had subsided and the anterior chamber returned to more nearly its normal depth. Failing to reduce the tension with miotics iridectomy should be resorted to at once, preceded by posterior sclerotomy.

Dr. BELL does not think well of posterior sclerotomy as a preliminary step in the performance of iridectomy for the relief of acute glaucoma because of the risk of vitreous hemorrhage, infection, and subsequent detachment of the retina. Dr. Bell said that he would not be surprised that we would some day find that the toxines produced by abscessed teeth, diseased tonsils, and disorders in the gastro-intestinal tract are responsible for a great many of the cases of glaucoma.

Dr. BRUDER referred to a case of glaucoma that had come under his observation in which the tension was subnormal. A few hours after the instillation of atropin it rose above normal and continued so until the effect of the atropin had been overcome.

Dr. H. W. WOOTTON presented a patient with unusual **retinal detachment**. The case was that of a man, 32 years of age, without history of trauma or constitutional disease. For the past six months he has complained of disturbed vision in the right eye. Previous to that he said that he had been disturbed for a long time by what appeared to be floating clouds before the field of vision of this eye. When first seen vision was reduced to fingers at two feet; field of vision contracted to the upper and outer quadrant. The ophthalmoscopic picture was that of a flat wavy membrane resembling a retina that had been torn from the ora serrata and extending from above downward in an oblique direction in the temporal half of the pupillary space. In as much as the fundus showed a normal red reflex to the outer side of the membrane there was a question as to whether or not the mass was a proliferating retinitis.

Dr. WOLFF thought that the retina had been torn from the temporal side.

Dr. BRUDER said that he could see vessels on both sides of the membrane and that therefore it must be a proliferating retinitis.

In Dr. BELL's opinion the retina was torn and folded upon itself.

Dr. WOOTTON presented a case of **retinitis proliferans in a diabetic subject**. In addition to the diabetes there was also a chronic interstitial nephritis and arteriosclerosis. The patient, 53 years of age, has been under treatment for diabetes for the past five years. Her vision began to fail in the left eye five months ago. Until that time she was not aware that vision in her right eye was defective. When first seen by Dr. Wootton her vision was reduced to light perception. Fundus examination revealed a grayish white mass at the site of the disk, protruding 2mm into the vitreous, somewhat larger than the disk in diameter. The left eye presented a typical picture of diabetic retinitis.

Dr. Wootton interpreted the picture in the right eye as a proliferating retinitis of a high degree involving mainly the papilla. The surface appeared uneven and the margin irregular, due to prolongation of connective tissue. The veins were enlarged and tortuous and the arteries contracted. Two small hemorrhages bordered the upper surface.

DISCUSSION: Dr. COHEN said that the case was unquestionably one of proliferating retinitis following a hemorrhage. He said that thrombosis of the central vein usually resulted in optic atrophy. In this case atrophy does not exist.

Dr. TENNER asked the speaker if he did not consider retinitis proliferans always due to hemorrhage. Dr. WOOTTON replied that he did.

Dr. F. N. IRWIN presented an unusual case of **scleritis, of possible tuberculous origin**. The present attack has existed for one month. Patient states that several years ago the same eye had been inflamed and she was then given injections. The eye had also been inflamed in infancy. The Wassermann reaction is negative, likewise Von Pirquet. Dr. Irwin has removed a piece of the tissue and injected it into a rabbit. He will report results at the next meeting.



DISCUSSION: Dr. SHERMAN said that he had had several cases of this type which he had treated with tuberculin. He was skeptical as regards its virtue but the good results obtained by others convinced him of its value.

Dr. GEORGE H. BELL showed a case of **sarcoma of the orbit** and exhibited X-ray plates of same. The case was far advanced and no operative interference was advised. He proposes to try radium.

Dr. BELL also presented a case of **plastic iritis** in both eyes due to severe mouth infection. When first seen both pupils were contracted and the aqueous cloudy. There was severe pain in both eyes, considerable exudation in the left pupillary space and marked ciliary injection. Wassermann test was +++ positive. The usual treatment for iritis was begun, including daily inunctions of mercury. In as much as the patient's teeth were badly decayed, fourteen were extracted during his first seven days' treatment at the hospital. Improvement in his eye condition was so rapid that Dr. Bell decided to discontinue the mercurial inunctions. The patient made a rapid recovery, leaving the hospital at the end of three weeks. He has now, slightly over nine weeks since treatment was begun, gained fourteen pounds in weight, his eyes are perfectly quiet, and his vision is  $\frac{20}{20}$  in each eye. He was given only seven inunctions of mercury. Dr. Bell thinks that unquestionably the condition of the teeth in this case was responsible for the iritis. He proposes to treat the patient for constitutional syphilis later.

DISCUSSION: Dr. SCHOENBERG said that the case was a very interesting illustration of how difficult it is at times to make a diagnosis of a non-syphilitic condition in a patient with syphilis. If the treatment had consisted only of the extraction of the teeth Dr. Bell's diagnosis could not have been disputed. But this patient received several inunctions of mercury before his decayed teeth were removed. The fact that the iritis began to improve after the mercurial inunctions were discontinued does not prove anything, since it is a well established fact that mercury, when absorbed, remains in the system for months and even years, during which time it continues its effect.

Dr. DWYER said that it is possible to recognize different

types of iritis clinically and that he had no doubt but that Dr. Bell's case was one due to mouth infection. He said that recovery was too rapid in this case for it to be classified as syphilitic.

Dr. HUBBARD referred to a case of **kerato-iritis** which recovered very rapidly after the extraction of several offending teeth.

Dr. ALGER thinks we may be too optimistic in regard to this new discovery but reported the case of a man who had had more than 100 attacks of iritis during the past thirty years. He had had every form of examination and treatment without result. During the last attack, two years ago, his teeth were found to be bad, those diseased were removed, the iritis subsided much more quickly than ever before, and there has been no return since.

Dr. BELL said that, according to his observation, it was not reasonable to assume that seven inunctions of mercury would cure a plastic iritis of specific origin.

Dr. BEN WITT KEY reported a case of **pulsating exophthalmos**. J. O., aged 49, admitted to the hospital Oct. 26, 1917, with history of a severe blow with a club on right side of head two months before. Five days after the injury he noticed slight protrusion of his left eye, which has gradually become worse; he is also conscious of a pulsation and noise on left side of his head. On inspection a definite pulsation of the globe synchronous with each heart pulsation could be seen. A well-defined bruit could be heard. X-ray plate gave no evidence of injury. Digital pressure caused the pulsation to stop. At the end of two weeks the patient was discharged from the hospital, the exophthalmos being greatly diminished and the pulsation very feeble.

## BOOK REVIEWS

**I.—The Indian Operation of Couching for Cataract.** Incorporating the Hunterian Lectures, Feb. 19 and 21, 1917. By Lieut.-Col. R. H. ELLIOT. With 45 illustrations, pp. 94. H. K. Lewis, London, 1917. 7/6 net.

The couching of cataract is one of the oldest operations in surgery which has gradually given way to better procedures, except in India where its popularity continues, particularly owing to social and religious conditions, though thanks to the efficient members of the I. M. S. the newer procedures are gaining steadily. As the operation of couching is still recommended under exceptional conditions by well-known operators in the West, Colonel Elliot's book arouses practical as well as historical interest. The author distinguishes between two methods of couching, depression and reclination; in the first the lens is pushed straight into the vitreous, while in the second, the later method dating from 1785, the lens is reclined backward, remaining attached by the suspensory ligament below. The site of approach is either anterior or posterior, as the puncture is made with a needle-like instrument through the aqueous chamber or posterior to the ciliary body.

The results of this operation as practiced by the native coucher, Colonel Elliot, from a study of many cases (780), finds to be as follows: quite a number of eyes are lost from panophthalmitis and iridocyclitis. Cases were usually seen from 1 to 10 years following the couching. The results are universally bad, 60% are lost. The vision in only 10% was  $\frac{1}{2}$  or better when the patients came under observation, the shorter the period after operation the better the sight. The causes of failure in the total cases were iridocyclitis 35%,

glaucoma 11%, imperfect dislocation of lens 9%, retina detached 1.5%, optic atrophy 2.5%, retinal pigmentation 0.5%, chorioretinitis 1.1½%. The iridocyclitis and glaucoma generally come on within a few days of operation. Detachment of the retina probably occurs in a greater percentage. As for the question whether the lens in the vitreous brings about retinal changes, as has been advanced by some, Elliot cannot find evidence to support this view, though he has found an undue distinctness of the choroidal vessels.

Fifty-four globes were available for microscopic examination. The cataractous lens was usually found floating freely in the vitreous or it was bound down to the ciliary body by exudate. In four the lens was displaced into the anterior chamber. The eyes lost by panophthalmitis were eviscerated and not available for examination. The evidences of iridocyclitis were frequent and plastic in character. The author's observation on changes in the vitreous are important. The vitreous was found detached and shrunken, with evidences of infiltration and of organization. The vitreous exudates under the microscope proved to be of the nature of inflammatory exudation, more or less structureless masses containing blood-cells and leucocytes; the anterior hyaloid membrane was definitely thickened and infiltrated. The optic nerve-head was usually inflamed and the author regards this as proving the existence of a lymph current in the vitreous body. Detachment of the retina occurred in 70% of these eyeballs, undoubtedly secondary to the vitreous changes. Glaucoma was present in nineteen eyeballs, the cause was always one of those accepted for secondary glaucoma.

The diagnosis of a couched cataract is next taken up and the question of what is to be done with these cases comes up for consideration. As a rule operators shrink from operating on these cases, and Elliot would urge that only the freely movable cataracts should be touched. In 1912 the author reported on the results of 18 cases in which the lens was removed; in 12 vision was improved, in 4 no change, and 2 were made worse, though he fears that the improvement was not lasting. It is fortunate for us that Colonel Elliot was interested in this remarkable operation and took advantage of the opportunities in India of collecting material and obser-

vations to write this complete and scientific study of the subject. While we in the West rarely see the results of the couching of cataract, the future of eyes in which lenses have become dislocated into the vitreous is important.

A. K.

II.—**Congenital Word-Blindness.** By Dr. JAMES HINSHELWOOD, Glasgow, pp. 112, published by H. K. Lewis & Co., Ltd., London, 1917. Price 4/net.

The studies and observations of the writer on word blindness, a subject to which he was the first to draw attention, are now collected in one volume which will be of great service to those dealing with congenital defects of childhood, particularly from its educational side. Word blindness means that with normal vision for letters and words the patient cannot interpret printed or written language.

In the first chapter the subject of acquired word-blindness is reviewed, as a knowledge of this condition is necessary to explain the congenital condition. The case history of a patient is reported whom the author was able to follow for many years after the onset of the cerebral lesion, and on whom an autopsy was obtained. This patient lost his visual memory for printed characters though he was able to read numbers and write to dictation. The only other cerebral defect was a right lateral homonymous hemianopsia. At autopsy a subcortical lesion was found causing isolation of the visual memory center, leaving this center intact.

The place where visual impressions are stored is the visual memory center, situated in the angular gyrus on the left side in right-handed people. Visual memory, the author has shown, can be furthermore subdivided into functions whose loss produce letter, word, and mind blindness. It is also to be remembered that there are independent visual memories for words and letters, for numbers and for musical notes, of which a single group alone can be lost. While these require mental attention and concentration, the author speaks of another group of visual memory for objects, persons, places, etc., which may be stored up in the visual memory centres of both hemispheres. Cases have been reported showing that there are partial forms of word blindness where an individual becomes

word blind to only one language. This suggests that our visual impressions are arranged in definite and ordered groups.

The visual memory center in the left angular convolution is connected with both occipital visual centers. If the visual memory center is destroyed by a cortical lesion, the word-blindness is accompanied by *agraphia*; a subcortical lesion, on the other hand, leaves the center isolated and cut off from other centers and communicating fibers, but the power to write is retained.

Cases of acquired word-blindness occasionally recover. When this occurs, improvement begins early. In those cases where the condition is permanent, something can be done by re-education. Hinshelwood believes that in these cases the corresponding center on the opposite side of the brain is educated, a procedure which has been noted in *aphasia*.

Congenital word-blindness is a defect in otherwise normal children, characterized by difficulty in learning to read, though they do remarkably well in oral instruction. The condition in the author's opinion is by no means so rare, and its proper recognition is important, as these children are often erroneously neglected or punished.

Twelve cases are reported, all showing the same defect in ability to recognize print or writing but with all the other cerebral powers unaffected. In only three did the defect include the non-recognition of figures, and all of them learned to write. One case made good progress in music. Hinshelwood finds that this condition preponderates greatly in boys, —of the twelve, ten were males. Congenital word-blindness is sometimes hereditary. An instance is reported where of a family of eleven, four of the boys had the greatest difficulty to learn to read; in the succeeding generation two boys of an unaffected mother showed this peculiarity. In these cases the cause is regarded as defective development. The *hemianopsia* so frequently associated with acquired word-blindness due to disease is never met with in the congenital variety.

In diagnosis the author particularly insists on "the gravity of the defect but also on the purity of the symptoms." The cases must not be confounded with those of general failure of cerebral development. The general intelligence of these

children is quite up to the average, and their auditory memory is intact.

As to treatment, these children can be taught to read with proper treatment and great perseverance. The author recommends personal instruction alone, and not in a class, and the use of the old-fashioned method of learning to read as the best and quickest. We are all greatly indebted to Dr. Hinshelwood for his researches in this important subject, as cases with this defect are first brought to the oculist for examination and the diagnosis, as the author has well shown, is not difficult.

A. K.

**III.—Consumption and Squint.** One of three clinical studies in Tuberculous Predisposition. By W. C. RIVERS, M.R.C.S., L.R.C.P., D.P.H. Published by Geo. Allen & Unwin, London, 1917.

The author found six squinting consumptives in five hundred examined, or about 1.2 per cent. This seeming excessive, he examined nine hundred controls and found four who squinted, or about 0.4 per cent., from which he concluded that concomitant squint is two or three times as common in consumptives as in the general population. During the examination of 10,239 children (non-tubercular) Hansen found an average of 2.5 per cent. with strabismus, which shows a wide variation with the average of the author's controls.

As to the inferences one is entitled to draw as to a causal connection between strabismus and phthisis, he states that it is impossible to conceive how any simple ocular defect short of blindness could of itself favor the development of pulmonary tuberculosis, even in the most indirect manner. As the squint long preceded clinical manifestation of tuberculosis, it is unlikely to have been caused by that disease in any declared form. For the causal sequence to happen, necessary postulates are: early tuberculous infection, early interference with one single nervous function (the fusion faculty of vision or kindred function), and then complete latency for some—perhaps thirty—years. He says that squint appears to be sometimes a mark of mental deficiency or a tendency, personal or familial, to certain bodily anomalies or malformations. It probably instances the presence of predisposition to tubercle (clinical

pulmonary tubercle) without itself forming an active constituent of a phthisical diathesis. Such active constituent may be some associated malformation of which squint is the stigma. He finally concludes "that according to his findings on his evidence there is undoubted association between concomitant strabismus and consumption, of predisposition to which latter disease squint forms a sign."

While the author's observations are interesting, and it is conceded that strabismus may be associated with tuberculosis as with other diseases, many would hesitate to accept his deductions on account of the insufficiency of data, the omission of consideration of the individual ocular conditions, and the wide variance in his control ratios with those of other reliable observers.

H. H. T.

**IV.—Medical Ophthalmology.** By ARNOLD KNAPP, M.D. Being one volume of "An International System of Ophthalmic Practice," edited by WALTER L. PYLE, M.D. Philadelphia, P. Blakiston's Son & Co., 1918. Octavo, pp. 509, with 32 illustrations. Price \$4.00.

There has been lacking hitherto a compact, systematic, well-indexed, single volume of reference in English devoted to the ocular symptoms of general medical diseases, hence the appearance of this book is very welcome.

The first section, consisting of 80 pages, written in collaboration with Prof. Marburg of Vienna, deals with the anatomy and physiology of the visual apparatus, and is illustrated with about thirty well-chosen diagrams. The next section, of 120 pages, treats of diseases of the nervous system, and following this come sections on diseases of the glands with internal secretion, diseases of the skin, circulation, and kidneys, digestive tracts, diseases of infectious nature and poisons.

The writer has drawn largely for his material upon the recognized German and French handbooks on these subjects and upon the transactions of the Ophthalmological Society of the United Kingdom. A captious reviewer might wish that he had exercised greater critical faculty in selecting and digesting some of his material; and that he had rejected some of the errors that are copied from one text-book to another. The



sections on syphilis, tuberculosis, and the nasal accessory cavities seem particularly valuable, since in them the writer's large personal experience has guided him to a great extent.

The volume will doubtless find its way into many hands and prove to be a very satisfactory book of reference.

W. A. H.

MISCELLANEOUS NOTES.

OPHTHALMOLOGICAL SOCIETY OF THE UNITED  
KINGDOM.

PRESIDENT: E. TREACHER COLLINS, F.R.C.S.

ANNUAL CONGRESS, 1918.

The next **Annual Congress** of the Society will be held on  
**Thursday, Friday, and Saturday, 2d, 3d, and 4th of May,**  
**1918.**

The arrangements will be as follows:

**THURSDAY, MAY 2d.**

(At the Royal Society of Medicine, 1 Wimpole Street, W.)

**MORNING.**

10 a. m. to 12.30 p.m.... Papers.

**AFTERNOON.**

2.30 p.m..... Discussion on "Plastic Surgery of  
the Eyelids." To be opened by  
Major H. D. GILLIES, Messrs. C.  
HIGGENS, and T. HARRISON  
BUTLER.

Members are invited to exhibit  
illustrative cases.

5 p.m..... Tea.

5.30 p.m..... Business Meeting.

Members will dine together in  
the evening.

**FRIDAY, MAY 3d.**

**MORNING.**

**Visit to the Metropolitan Asylums Board Ophthalmia School, Swanley.**

Discussion on "Contagious Diseases of the Conjunctiva." To be opened by Messrs. J. B. STORY, SYDNEY STEPHENSON, Major J. F. CUNNINGHAM, and Capt. J. WHARTON.

**AFTERNOON.**

3.30 p. m. . . . . **Visit to Museum of Royal College of Surgeons.**

Exhibition of Specimens illustrating War Injuries.

Demonstration of Specimens of Eye Injuries by Col. W. T. LISTER.

**EVENING.**

**At the Royal Society of Medicine, 1 Wimpole Street, W.**

8 p.m. . . . . Exhibition of Cases.

8.30 p.m. . . . . Papers.

**SATURDAY, MAY 4th.**

**MORNING.**

**At the National Hospital for Paralysis, Queen's Square.**

10 a.m. . . . . Clinical Meeting.

Members are invited to show Ophthalmological Cases of Neurological Interest.

Members desirous of reading papers, showing cases, or taking part in the discussions, are requested to communicate as soon as possible with Mr. M. S. MAYOU, 30 Cavendish Square, London, W. 1.

Papers and communications, subject to the judgment of the Council, will be printed in full in the "Transactions."

Under the Bye-laws readers of papers must not exceed

twenty minutes, subsequent speakers ten minutes. The openers of the discussions are allowed twenty minutes.

*All communications must be type-written.*

A Museum will be held at the Royal Society of Medicine, with a Special Exhibition of Perimeters. Members wishing to exhibit drawings, specimens, or instruments should communicate with Mr. A. C. HUDSON, 50 Queen Anne Street, London, W. 1.

S. A. KINNIER WILSON } *Hon. Secs.*  
M. S. MAYOU }

### THE AMERICAN JOURNAL OF OPHTHALMOLOGY

The first number of the new *American Journal of Ophthalmology* has been published, and the editors are to be complimented on its extremely creditable appearance. The journal is the result of the amalgamation of the *American Journal of Ophthalmology*, the *Annals of Ophthalmology*, the *Ophthalmic Record*, *Anales de Oftalmologia*, *Ophthalmology*, *Ophthalmic Year Book and Literature*. The editorial staff consists of Edward Jackson, editor, Clarence Loeb, associate editor, A. Alt, M. Uribe-Troncoso, M. Wiener, C. A. Wood, and H. V. Würdemann. There are many American and foreign collaborators. The journal will appear monthly, published by the Ophthalmic Publishing Co., 7 W. Madison Street, Chicago, Ill. The annual subscription price is \$10.

The number of nearly 100 pages is wide in its scope, and comprises nine original and well-illustrated papers, several translations from foreign journals, short abstracts, society proceedings from Philadelphia, Colorado, and Chicago, editorials, book notices, news items, index of ophthalmic literature, and digest of the literature. The last part, the digest of the literature, is paged separately and ultimately will form, when bound together, the *Ophthalmic Year Book*. The size of the page in the new journal, the quality of the paper, and the type are all excellent; the lines are perhaps too close for comfortable reading.

The ARCHIVES welcomes the new journal with every good wish for its success.

# ARCHIVES OF OPHTHALMOLOGY.

## THE EYE IN NERVOUS DISEASE.

AN INTRODUCTION TO NEUROLOGICAL OPHTHALMOLOGY,  
PREPARED FOR THE N. Y. NEURO-SURGICAL SCHOOL  
FOR ARMY MEDICAL OFFICERS.

By WARD A. HOLDEN, M.D.

(With three figures on Text-Plates XI.-XIII.)

**I**N the routine examination of the eyes of a patient with nervous disease or injury, we wish to determine particularly the following points:

- (a) The sensibility of the cornea.
- (b) The size, shape, and reactions of the pupils.
- (c) Any abnormalities of the extrinsic ocular muscles—  
nystagmus, spasm, squint, or paralysis.
- (d) The acuteness of distant and near vision and the  
fields of vision.
- (e) The condition of the optic disc as respects atrophy,  
edema, or alterations in the blood-vessels.
- (f) Other anomalies such as exophthalmos, retraction  
of the eyelids, and evidences of existing or previous  
inflammation, injury, or operation.

The methods of conducting these examinations and the neurological significance of the results obtained will be taken up seriatim.

### CORNEA.

The *sensibility* of the cornea is tested most conveniently by touching the cornea with a bit of absorbent cotton twisted

into a thread. Since certain patients shrink from this test, it is well to touch the cheek first and ask the patient if he feels the cotton. Less objection will then be made to touching the cornea itself. In normal conditions when the cornea is touched the patient will wink and also give evidence of feeling some discomfort. The neurotic patient may jerk away as if he had been burned with a hot iron, but this reaction is due more to fear than to actual pain.

In other cases the winking is slow or absent, indicating hypesthesia or anesthesia of the cornea. After section of the ophthalmic division of the fifth nerve, in some cases of herpes zoster of this branch, and after the instillation of cocaine, there is absolute anesthesia of one cornea and touching it causes no winking. Again there may be merely diminished sensibility of one or both corneas. If unilateral, this hypesthesia, without facial hypesthesia, may mean a tumor of the cerebellum on the same side. In such cases there will be also coarse nystagmus to the same side, unstable station, and muscular weakness with a tendency to fall to the same side. Or, hypesthesia of one or both corneas may be a sign of hysteria, when corroborating symptoms may be found, such as anesthesia of the pharynx or other parts, a history of aphonia, or contraction of the fields of vision with normal pupils and optic discs.

Diminished sensibility of one cornea, therefore, means a gross lesion of the fifth nerve of that side, a lesion of the cerebellum of that side compressing the fibers of the fifth nerve, or hysteria.

#### THE PUPILS.

**The pupils in health.** The *width* of the pupils is greater in myopic individuals than in hyperopic, and greater in youth than in old age. In adolescence the average width of the pupils in moderate illumination with both eyes open is about  $4mm$ , one-third of the width of the cornea. In middle age the average is about  $3.5mm$ , while in old age it is  $3mm$  or less.

The *shape* of the pupils is round in adolescence, slightly irregular in middle life, and often considerably irregular in old age.

The *reactions* of the pupils are the sensory, the direct light, the consensual light, and the convergence reaction.

The *sensory* reaction consists in a dilatation of the pupils when, for example, the skin of the temple is scratched or the skin of the neck is pinched. While it is usually well marked in early life, in middle age it is inconstant, hence its absence is of very slight diagnostic importance.

The *direct* reaction is the contraction that takes place when light is thrown into the eye observed, and the *consensual* is the contraction that takes place in one eye when light is thrown into the other.

In health, the two pupils are always of equal size, and light thrown into one eye causes a moderate contraction of both pupils, while light thrown into both eyes causes an excessive contraction of both pupils.

The *convergence* reaction is the contraction which occurs when the eyes are brought from distant fixation into convergence and accommodation for near fixation.

The light reactions are well marked in early life, less marked in middle age, and often rather sluggish in old age when the blood-vessels are sclerosed. The convergence reaction is well marked at all ages.

The *tests of pupillary reaction* may be conveniently made by having the patient sit facing a window at a distance of several feet. If he is then directed to look up at the ceiling, we displace the annoying corneal reflex which ordinarily overlies the pupil and renders it a difficult matter to determine its shape. The eyes are now covered alternately to obtain the consensual light reaction, and next, while one eye is covered the other is alternately covered and exposed to obtain the direct light reaction. The convergence reaction is obtained by directing the patient to fix the observer's finger held near the eyes in line with the point on the ceiling at which he has been gazing.

A finer test that will reveal slight sluggishness is that of throwing light into the eye from a distance with the ordinary ophthalmoscope. The electric ophthalmoscope is unsuited to this test. A common test is that of throwing light into the eye with a pocket electric flash light. This is, however, a coarse test that will fail to discover many sluggish pupils

unless the light is held at some distance from the eye or so held that its rays enter the eye obliquely in order that few of the rays may fall upon the center of the retina.

**The pupils in disease.** Before considering anomalies of the pupil it is well to call to mind the nerve supply of the iris. The sphincter muscle is supplied by a branch of the 3d nerve which, when stimulated, causes a contraction of the pupil, and when inhibited, causes a moderate dilatation. The radiating arteries of the iris, on the other hand, have their muscular coats supplied by fibers of the sympathetic nerve which, when excited, cause a dilatation of the pupil and, when inhibited, lead to its contraction.

*Sympathetic pupillary disturbances:* Excitation of the cervical sympathetic causes a dilatation of one or both pupils without visual disturbances and often without the knowledge of the patient. The dilatation is usually transitory and occurs in the neurotic individual, the menstruating woman, or the young girl, following excitement, fatigue, or emotional stress. A more lasting and sometimes permanent disturbance is the contraction of the pupil, usually unilateral, that follows compression of the sympathetic fibers or center.

An injury of the spinal cord involving the cilio-spinal center which lies at the level of the lower cervical and first dorsal vertebræ, or pressure on the sympathetic fibers by an enlarged bronchial gland in tuberculosis, or pressure by cervical glands, or a goiter, may produce a unilateral complex of symptoms consisting of contraction of the pupil, narrowing of the palpebral aperture, and slight sinking of the eyeball into the orbit. Here also there is no disturbance of vision and the patient may not be aware of anything wrong. In doubtful cases, the instillation of 4% cocaine solution will confirm the diagnosis, for cocaine in the normal eye excites the sympathetic nerves and causes dilatation of the pupil and widening of the palpebral aperture. In these cases of pupillary contraction from sympathetic paralysis, cocaine does not dilate the pupil or widen the palpebral aperture in the affected eye, while it has its usual effect in the other eye.

*Third nerve pupillary disturbances:* Meningeal irritation may cause a spasm of the sphincter muscles with contraction of both pupils. Compression of the nucleus of the nerve for



the sphincter muscle or of its fibers in the 3d-nerve trunk will cause dilatation of the pupil. But, since the nucleus for the sphincter muscle and that for the muscle of accommodation—the ciliary muscle—lie close together and apart from the nuclei for the other muscles supplied by the 3d nerve, and since the fibers for the sphincter and the ciliary muscles run close together in the 3d-nerve trunk, this paralytic dilatation of the pupil is usually accompanied by paralysis of the muscle of accommodation and the patient is unable to see near objects clearly. In these cases, too, the light reaction which is mediated by the 3d nerve is absent, and usually the convergence reaction as well. In the sympathetic pupillary disturbances, on the contrary, the light and convergence reactions are never abolished, although they may be somewhat reduced.

Of clinical interest are the following types:

- (a) Paralysis of accommodation in each eye with pupils of normal size and reaction often follows diphtheria and lasts a few weeks only.
- (b) Paralysis of accommodation and dilatation of the pupils with loss of light and convergence reactions in both eyes, without other muscular anomalies, occasionally occurs in ptomain poisoning and is likely to be permanent.
- (c) The same condition, known as ophthalmoplegia interna, occurring in one eye only and lasting a long time, is almost invariably due to a luetic nuclear lesion.

*Argyll-Robertson pupil.* Half a century ago, Professor Argyll-Robertson of Edinburgh described a condition in which the light reflex of the pupil is lost while the convergence reflex is preserved, and this he found to be associated with anomalies of the knee jerk, hence, with disease of the spinal cord.

This condition is to be explained by a break in the reflex arc for the light reaction. Fibers from the macular region of the retinas run in the optic nerves and tracts to the primary visual centers, the external geniculate bodies, chiefly, here connecting with the adjacent nuclei for the sphincter muscles of the irides. It is now generally agreed, although not anatomically proven, that the break in the arc must lie in the

fibers connecting the primary visual center with the sphincter nucleus. In support of this view is the fact that non-luetic tumors in the region of the third ventricle by pressure here produce typical Argyll-Robertson pupils.

Our views as to the causation of the Argyll-Robertson pupil, or reflex iridoplegia, and as to its onset and course have become very definite in recent years. Almost without exception it is a symptom of syphilis of the nervous system, and in its incipient stages it appears often within a few months after the initial infection in persons who are to exhibit frank symptoms of syphilis of the nervous system many years later. It begins with slight irregularity and inequality of the pupils, followed by sluggishness of the light reaction. In most cases the pupil gradually becomes contracted to a degree that can be explained by a spasm of the sphincter muscle only and the light reflex is entirely lost. Later, the pupils may become abnormally dilated—in some cases they are large throughout—and with this late dilatation, or before it, the convergence reaction may become sluggish. Vision is never affected by the onset of reflex iridoplegia alone. If near vision is affected, there is absolute iridoplegia with an accompanying paralysis of the muscle of accommodation, namely, an internal ophthalmoplegia.

A certain degree of sluggishness of the pupils, of a transitory nature, is met with in persons who are fatigued or under emotional strain. Hence a second examination is desirable after an interval of rest in such individuals.

#### EXTRINSIC MUSCLES OF THE EYE.

Nystagmus, spasm, concomitant strabismus, and paralysis are here to be considered.

*Nystagmus:* When nystagmus dates back to infancy it has no great diagnostic significance. Partial albinism or opacities of the media of the eye may be ocular causes, and difficult labor or hereditary lues may be cerebral causes, giving rise to nystagmus that is mostly constant, but increases when the eyes are moved from the primary position. A slight amount of nystagmus when the eyes are turned far to one

side is fairly constant in the neurotic and the fatigued and may be called physiological.

A well marked nystagmus acquired after infancy always has significance. This may come on with the most diverse intracranial conditions, but certain types have definite significance. First, there may be nystagmus when an ocular muscle is about to be paralyzed or when it is recovering from a paralysis. This nystagmoid twitching is in the direction of the action of the muscle affected and is usually more pronounced in the affected eye than in the other. Nystagmus on looking upward often indicates a midbrain growth and precedes a paralysis of the superior rectus. Marked nystagmus in looking to one side often means a pontine lesion and precedes an associated paralysis of ocular movements to that side. Nystagmus coarse and slow to one side and fine and rapid to the other usually means a cerebellar lesion on the side of the coarse movement. In many cases of disseminate sclerosis there is slight nystagmus in looking straight forward which becomes exaggerated when the eyes are turned in any direction. The matter of aural nystagmus is too considerable to be taken up here.

*Spasm:* Spasmodic contraction of the orbicularis muscle or of the internal recti producing diplopia is common in hysteria. Spasm of an internal rectus with contracted pupils is often a sign of cerebral irritation, and spasm causing conjugate deviation of the eyes is usually due to pontine irritation.

*Concomitant strabismus:* When of the convergent variety, this comes on in hyperopes about the age of two years, and when of the divergent variety, it comes on in adolescent myopes. There is here no limitation in the extent of mobility in either eye, but in one eye or both mobility is increased in one direction and diminished in the contrary direction. There is no diplopia. When one eye is blind or amblyopic and is not used for visual purposes, it is apt to turn inward in early life and in adult life outward.

*Paralysis:* Congenital paralysis due to hemorrhage at birth or malformation of muscles appears immediately after birth. The external rectus is the muscle most frequently involved and a differentiation between convergent strabismus due to a birth palsy of the external rectus or to hyperopia

depends often upon the immediate appearance of the former and the periodic development of the latter at about the age of two years. Next in frequency to birth paralysis of the external rectus is paralysis of one or both superior recti, sometimes accompanied by ptosis. Diplopia is not observed in these congenital cases.

Acquired paralysis of an extrinsic muscle of the eye is revealed to the patient by the existence of diplopia and to the physician by defective mobility of the eye in some direction. Tests for defective mobility, as for nystagmus, are best made by having the patient fix a bright object in the distance and then turn his head from side to side and up and down. This brings out the full excursions of the eyes to all directions and avoids the complication of convergence which comes into play when the patient fixes the finger or other near object.

If there is complete, or almost complete, paralysis of a rectus muscle, simple inspection will reveal the defect. With a slight paresis of a rectus muscle or of a superior oblique, it is necessary to cover each eye alternately, when the affected eye will be found to lag behind if moved in the direction in which the paretic muscle should move it. Thus, for example, with paresis of the right external rectus, there will be no movement of the eyes on alternate covering except when the eyes are turned far to the right when alternate covering will show that the right eye when covered is convergent. Similarly, with paresis of the right superior oblique muscle, the eyes will be balanced under alternate covering except when turned down and to the right, when the right eye if covered will be found to stand higher than the left and to be convergent. After a few weeks a paralytic condition may become complicated by contracture of the muscles antagonistic to the paralyzed muscle, when the status of the muscles must be rather laboriously worked out by holding a red glass before one eye and noting the relative position of the red and white double images of a distant light in all four quadrants of the field of fixation. This test had better be left to the trained ophthalmologist.

The position of the patient's head is often a guide to the muscle paralyzed, for if he does not close one eye to avoid

diplopia he will turn his head to the right and his eyes to the left with paralysis of the right external rectus, and will turn his head down and to the right and his eyes in the contrary direction with paralysis of the right superior oblique, thus increasing the range of mobility in which he sees single.

The *site of the lesion* in ocular palsies is almost exclusively in the nucleus of the supplying nerve or in its trunk. Facial palsy and pseudo-bulbar palsies are frequently due to a supra-nuclear lesion, but since the nuclei of the ocular muscles chiefly have a cortical representation in both hemispheres, practically the only paralyzes of supranuclear origin are an occasional ptosis and rarely a transitory conjugate deviation of the eyes.

In order of frequency of involvement, the sixth nerve with its long basilar course is first, then the third nerve in some or all of its branches, and finally the fourth nerve. The sixth-nerve trunk is frequently involved in hemorrhages and exudations at the base of the brain, and a paralysis of both external recti alone is almost necessarily basilar, while paralysis of muscles supplied by both third nerves is usually basilar. In cases of increased intracranial tension from tumor, a paralysis of one external rectus, while the other cranial nerves are spared, is quite common, but this has no great localizing value since it seems to be caused frequently by compression of the nerve trunk by a cerebellar artery (Cushing) and is a sign merely of general increased intracranial pressure.

Destruction of the nucleus of the sixth nerve has more complicated results, since it brings about a paralysis of associated movements to the same side. Disturbance of the middle frontal convolution will cause a spasm or paralysis of associated lateral movements. However, tracts of fibers run from this *cortical* center to the sixth-nerve nucleus which forms the *pontine* and chief center for associated lateral movements, through the association fibers which run in the posterior longitudinal fascicle from the sixth-nerve nucleus of one side to reach the nucleus for the internal rectus of the opposite side. Apoplexy may cause a transitory conjugate deviation from cortical or supra-nuclear pressure, but a permanent inability to turn the two eyes to the right or to the left means a lesion of the sixth-nerve nucleus of the same side. Although

associated lateral movements to one side are thus completely abolished, the affected internal rectus can from another innervation functionate to a considerable extent when efforts at convergence are made.

In cases of nuclear paralysis of the muscles supplied by the third nerve, very frequently the sphincter of the pupil and the muscle of accommodation are spared and the condition is then known as external ophthalmoplegia. When the intrinsic muscles are affected also, the condition is known as total ophthalmoplegia.

Nuclear lesions of the third nerve, as a rule, affect the recti muscles first and the levator of the upper lid later or not at all. Compression of the nerve trunk usually causes ptosis from the beginning and frequently the intrinsic muscles are affected as well as the extrinsic. Pressure on the third-nerve trunk where it lies near the cerebral peduncle produces third-nerve paralysis on the same side and hemiplegia of the opposite side. A lesion in the red nucleus produces a paralysis of the superior rectus on the same side and athetosis of the opposite side of the body.

There are no particular signs by which a nuclear can be distinguished from a trunk lesion of the fourth nerve, except that here, as in the case of other cranial nerves, a trunk lesion may be preceded by meningeal headaches, whereas nuclear lesions may come on without premonitory symptoms. It is not difficult to tell whether there has been an involvement of the fourth nerve in cases of external ophthalmoplegia, because when an effort is made to look down and out, the superior oblique rotates the eyeball slightly down and out if the fourth nerve is intact, while this movement is wanting if the fourth nerve is involved as well as the third.

*The acuteness and the fields of vision:* Most affections of the optic nerve cause diminution in the acuteness or in the fields of vision, and the nature and extent of recent visual deterioration are often of as great importance in diagnosis as inspection of the optic-nerve head with the ophthalmoscope.

*Tests of distant vision* should be made with the patient's distance glasses, if he has them, and tests for near vision with his reading glasses. If the vision for distance is below normal and he has no distance glasses one is sometimes compelled

to rely on his statements as to whether there has or has not been recent failure of distant vision.

In the early forties, one's near vision fails from presbyopia, so a history of failing vision in middle age or later is of no neurological significance unless the patient is sure that it is his distant vision that has failed. A careful determination of the refraction is often desirable. In testing the illiterate or the aphasic, the Fridenberg test card on which groups of squares graduated in size replace the usual letters is especially useful, since the illiterate will count the squares in the group indicated and the aphasic will hold up an equal number of fingers.

*Tests of the field of vision:* When vision is very poor, tests may have to be made with the pocket flash light or with the moving hand, but with fair vision for routine purposes a white pinhead of about 3mm is the most useful test object. With the normal eye the field for this 3mm test object has almost the same extent as that of the usual 1cm perimetric test object, but defects in the field of an abnormal eye are much more readily discovered with the smaller object. When this test object is held half way between the eye of the observer and that of the patient, 50cm apart, the field of each eye should be equal in extent and if in any meridian the patient loses sight of the test object before the observer, his field is restricted in that meridian. In testing for relative hemianopsia and for central scotomata, a small red test object is useful, or the same 3mm pinhead may be used by the observer while standing several feet from the patient who fixes the observer's eye. The greater the distance the more delicate the test.

Bjerrum devised a black cloth screen 2m in diameter to be viewed at a distance of 2m, and used test objects of white ranging from 1 to 10mm in diameter, mounted on black rods which are nearly invisible against the background. His method is useful for plotting small or relative defects such as central scotoma, enlargement of the blind spot, or the like. Small hand compimeters are rather more useful than perimeters in plotting defects near the point of fixation.

Considerable practice is required to plot a visual field accurately with the perimeter because the patient becomes

fatigued and unreliable in his answers, or may not maintain proper fixation. What we wish to know for diagnosis is not so much the actual amount of contraction as the type of contraction, and this can be determined by the simpler tests.

**Contraction of the field of vision**, in nervous diseases, is found chiefly in four types, namely, concentric contraction, central scotoma with normal limits of the field, bitemporal hemianopsia or loss of the temporal half of the field for each eye, and homonymous hemianopsia or loss of the right or of the left half of the field for each eye.

*Concentric contraction* is the rule in simple atrophy of the optic nerve such as that occurring in tabes. This same sort of field is found in beginning post-neuritic atrophy, such as that following the papilledema of brain tumor. The atrophy following papilledema is caused by the compression of the nerve fibers, and the arteries as well, by exudated material, and in its final stages we find the nasal half of the field defective, just as the nasal half of the field fails in the compression atrophy of the nerve occurring in glaucoma from high intra-ocular tension. A concentric contraction of the field becoming smaller as the test is continued is known as the fatigue field and is a functional disturbance often associated with hypæsthesia of the cornea.

*Central scotoma* is chiefly due to affections of the papillomacular bundle—the group of visual neurones beginning in the ganglion cells of the macular region of the retina, passing to the temporal sector of the optic-nerve head and then extending back through the optic nerve as its central or axial bundle of fibers. This bundle is particularly susceptible to compression and to poisons. Central scotoma in one eye due to pressure is found with frontal-lobe tumors of the same side, and also with retained secretion in the posterior ethmoid cells in acute sinusitis. Central scotoma of both eyes of toxic nature is seen in chronic tobacco poisoning, in acute wood-alcohol poisoning, and in diabetes. A large central scotoma, or a number of minute scotomata near the center, is the commonest visual-field defect in disseminated sclerosis, although concentric contraction is often found with or without the central scotoma. Furthermore, hemorrhage in the macular





## ILLUSTRATING DOCTOR HOLDEN'S ARTICLE, "AN INTRODUCTION TO NEUROLOGICAL OPHTHALMOLOGY."

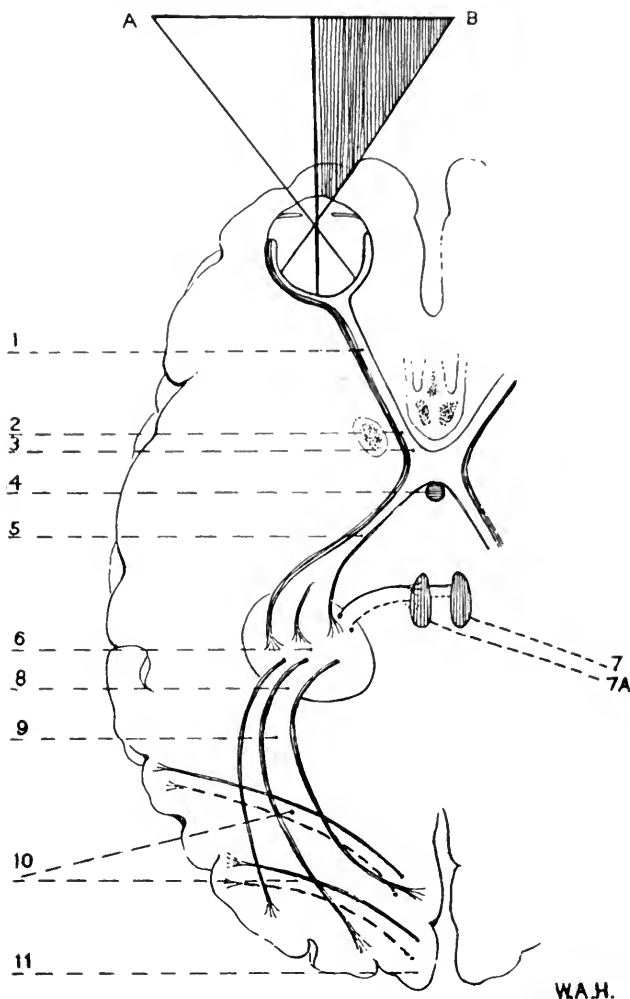


FIG. I.—A DIAGRAM OF THE LEFT VISUAL PATHWAY.

The dark outer portion of the optic tract and nerve represents the uncrossed fibers, which run from the temporal half of the retina and correspond to the shaded nasal half of the field B. The light, inner portion of the tract and nerve represents the fibers which, crossing in the chiasm, run from the nasal half of the retina and correspond to the unshaded temporal half of the visual field A.

1. *Optic nerve in the orbit.*

Pressure from hemorrhage, œdema, or tumor causes various degrees of amblyopia and various contractions of the field, with papilloœdema later, or with simple atrophy of the disk.

2. *Optic nerve in the optic foramen.*

Pressure here from diffuse periostitis following ethmoiditis causes first a central scotoma and later complete blindness, with papilloœdema some time after the onset. Pressure here from localized luetic periostitis causes first a peripheral defect in some portion of the field. Pressure here after fracture of the bone through the optic foramen causes very poor central vision and usually a lateral or vertical hemianopic defect in the field, with pallor of the disk.

3. *The optic nerve just anterior to the chiasm.*

Pressure here from tumor of the frontal lobe causes first central scotoma and later blindness with pallor of the disk.

4. *The infundibulum of the pituitary body.*

Dilatation of the infundibulum or enlargement of the pituitary body will compress the crossing fibers in the chiasm and cause defects in the temporal half of the field of each eye, with pallor of each disk, accompanied usually by a slight œdema of each disk.

5. *Left optic tract.*

Pressure here causes right homonymous hemianopsia with pallor of both optic disks.

6. *The external geniculate body, the anterior quadrigeminal body, and the optic thalamus,* in which terminate the first visual neurones coming from the retina. Association fibers run hence to the nuclei of the 3rd nerves, 7 and 7A. Disturbance of these association fibers causes reflex iridoplegia (Argyll-Robertson pupil).

8. The beginning of the second visual neurones, which make up the *Optic Radiation of Gratiolet*. A lesion here causes right homonymous hemianopsia with possible involvement of the nerves supplying the ocular muscles and also the auditory nerve. A lesion just posteriorly in the internal-capsule region may cause hemiplegia or hemianesthesia as well as hemianopsia, without pallor of the optic disks (*Carrefour sensitif* of Charcot).

9. Pressure upon the optic radiation here may cause right homonymous hemianopsia without other symptoms.

10. *The association tracts running from the calcarine fissure to the angular gyrus.* A lesion here will cause hemianopsia and, if on the left side, alexia or agraphia as well. Destruction of the angular gyri and neighboring structures in both hemispheres, even with intact optic radiations, will cause complete mind blindness.

11. *The pole of the occipital lobe,* the center for central vision. Destruction of the pole of each occipital lobe will cause a central scotoma in the field of each eye.

region of the retina in cases of arterial sclerosis, causing central scotoma, is quite common.

*Bitemporal hemianopsia* is caused by pressure upon the crossing fibers in the optic chiasm and is usually due to an enlarged pituitary body or a dilated infundibulum compressing the chiasma from behind, below or above. In the beginning, there is a relative loss of the temporal half of each field—that is, large test objects are recognized, but small white or colored test objects are not seen. Later, not even a light can be recognized in the temporal fields and the upper or lower nasal quadrants also become involved. Finally, the fields may be lost entirely and complete blindness in both eyes result. A radiograph of the sella turcica will usually show enlargement or distortion of the sella.

*Homonymous hemianopsia* can be caused by disturbance of the visual fibers at any point between the chiasm and the cortex of the cuneus. (See Fig. 1.) The lesion occurs frequently in the internal capsule or in the cortex about the calcarine fissure, but is quite rare in the optic tract. In cases of tract lesion, the optic discs become pale and accompanying basilar signs usually render the diagnosis possible. In lesions behind the optic thalamus, the optic discs remain normal. The so-called hemianopic loss of pupillary reaction when light is thrown upon the blind half of the retina, as a diagnostic sign of tract hemianopsia, is of no value whatsoever and should no longer be described in the text-books.

When the lesion causing the hemianopsia involves the corpora quadrigemina, paralysis of ocular muscles and deafness are likely to be present. A lesion in the optic radiation, if near the thalamus and internal capsule, frequently is accompanied by hemiplegia of the same side and less frequently by hemianesthesia. Lesions further back, subcortical or cortical, may cause hemianopsia without other symptoms if the association tracts are not involved. These association tracts run from the visual perceptive center in the calcarine region on the mesial surface of the occipital lobe to the visuo-psychic sphere which extends on the convexity of the lobe as far forward as the angular gyri. Destruction of these higher centers on both sides will lead to mind blindness. Destruction of the visual fibers of the optic radiation, together with certain

association tracts on the left side, will cause hemianopsia with alexia or agraphia.

It has been wrongly assumed by some that a so-called hemiachromatopsia indicates an involvement of a special color center. Hemianopsia, however, may range in degree from a slight relative to an absolute defect, and when the field is complete for large white objects but defective for one or more colors, it will be found that the field is equally defective for such small white objects as have a field equal to that for each color in the normal eye, as was pointed out by the writer many years ago.

*Gunshot injuries of the occipital lobe* producing hemianopsia have been very frequent in recent wars and the records of army surgeons have contributed not only to our knowledge of the clinical varieties of the hemianopic conditions but also in greater degree to our knowledge of retinal representation in the cortical visual area—the area striata. A paper on “Disturbances of Vision from Cerebral Lesions,” in *Brain*, 1916, p. 37, by Gordon Holmes and W. G. Lister, summarizing their records made in base hospitals in France, is a contribution of very great value. They group their cases of visual-field defects into four categories:

I. Quadrantic symmetrical defects in the two fields, (a) one quadrant wanting, (b) two quadrants wanting, and (c) three quadrants wanting. The inferior quadrants were wanting much oftener than the superior, because a superior defect means a lesion in the inferior portion of the area striata, which is likely to involve the cerebellum and be fatal.

II. Central scotoma in the field for each eye, usually symmetrical, due to injury of the tip of each occipital lobe.

III. Homonymous hemianopsia with central scotoma (the central scotoma representing a central or paracentral hemianopic defect on the side opposite to the complete hemianopsia) due to an oblique injury through the optic radiation on one side and the posterior tip of the occipital lobe on the other. (See Fig. 2.)

IV. Paracentral symmetrical homonymous scotomata, sparing central vision and being more extensive the further forward the injury.

These authors conclude, in brief, that each macula lutea

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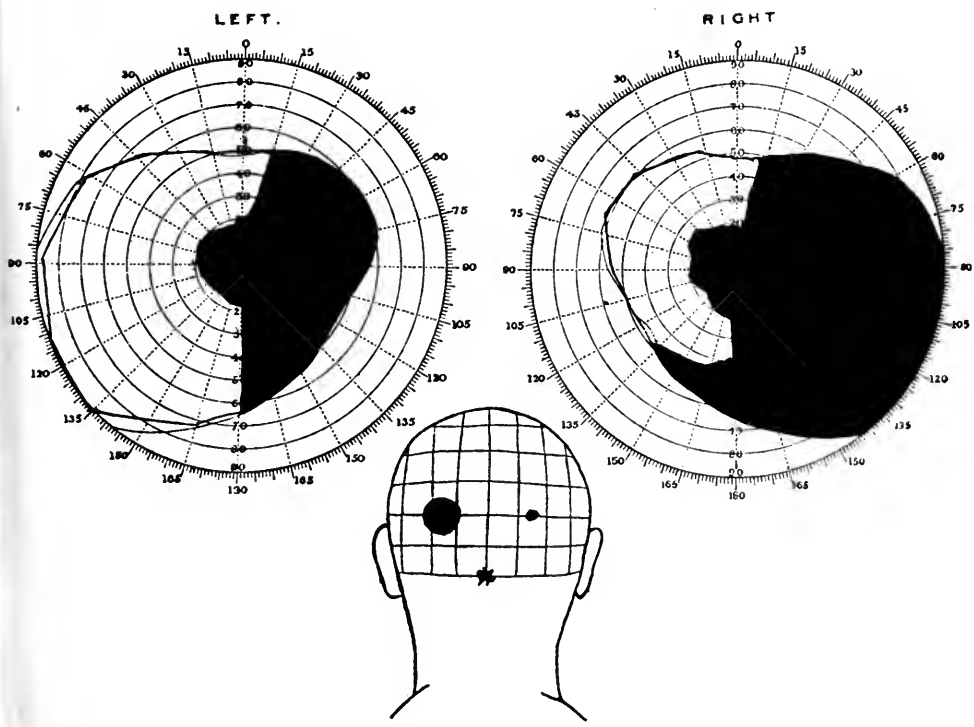


FIG. 2—After Gordon Holmes and W. T. Lister. Rifle-shot wound of both occipital lobes; entry on the left, exit on the right; producing almost complete right homonymous hemianopsia, and also symmetrical central defects in the left halves of the field extending 20° from the fixation point in each meridian.



has a representation in one occipital lobe only, not in both, as had generally been believed; that the center for the macula lies in the posterior extremity of the area striata, probably on the margins and the lateral surface of the occipital poles; that the upper portion of the retina (and the lower portion of the field of vision) has its representation in the cortex above the calcarine fissure and the lower portion of the retina in the cortex below the calcarine fissure; and that the representation of the portion of the retina near the macula lies near the pole of the occipital lobe, while that of the periphery of the retina lies anteriorly.

It has long been recognized that when a right homonymous hemianopsia, for example, was later followed by a left homonymous hemianopsia, due to a second plugging of an artery, a small central field was sometimes preserved. These authors explain such cases by calling attention to the fact that the tip of the occipital lobe is often supplied by the posterior cerebral artery, while the rest of the area striata is supplied by the middle cerebral artery. Thus, plugging of the branches of each middle cerebral artery would produce bilateral homonymous hemianopsia, but might spare central vision in each eye, since the cortical centres for central vision might be supplied by the posterior cerebral arteries.

#### OPTIC DISC.

The ophthalmoscopic changes in the optic-nerve head that interest us are atrophies and edemas. The *simple atrophy* or primary degeneration of the optic nerve is in the great majority of cases a symptom, early or late, of tabes. The retinal neuron first loses its medullary sheath, and later the axis cylinder and ganglion cell are absorbed. The spaces left are partly filled by proliferated neuroglia and the small blood-vessels disappear. In consequence the entire nerve head loses its pinkish hue and grows gradually paler until finally it becomes glistening white and sharply outlined, while the retinal arteries, whose function is to nourish the ganglion-cell and nerve-fiber layers of the retina, become narrow and sclerotic.

The patient notices first a failure of acuteness of vision,

and tests of the field show peripheric contraction. Central and peripheral vision fail gradually together and after the lapse of months or years complete blindness ensues, usually in both eyes.

Pressure upon one optic tract causes a slight general pallor of each disc, as does pressure upon the chiasm by an enlarged pituitary body. In the latter case we encounter a very slight edema of the disc as well. In optic-nerve affections due to ethmoid disease and to wood-alcohol poisoning, there may be at first considerable edema of the nerve head which soon passes off and is followed by the ophthalmoscopic picture of simple atrophy of the optic disc.

When the *papillo-macular bundle* alone is involved, causing a central scotoma without peripheric contraction of the visual field, the inferotemporal third of the optic disc becomes pale while the remainder of the disc retains its pinkish hue. This is the condition found in nicotine poisoning, diabetes, and in most of the optic-nerve involvements of disseminated sclerosis.

*Papilledema*, as it occurs in cases of brain tumor, is now generally regarded as being of a purely mechanical character, coming on in both eyes about the same time and running a fairly similar course in the two. A papilledema almost similar in character, but usually with less elevation of the disc and with more extensive involvement of the retina, occurs with meningitis. This variety is of a more inflammatory nature and the eye on the affected side of the head is involved before the other. The starting point of papilledema in brain tumor is a blocking of the passage from the third ventricle to the fourth. Blocking of this passage leads to an over-accumulation of liquid in the third and lateral ventricles, and this excess of liquid compresses the soft brain matter and forces the liquid in the subdural space out into the subdural lymph space of the optic-nerve sheaths which are continuous with the other.

The early or late appearance of papilledema depends much upon the situation of the tumor. A tumor of the third ventricle may block the passage to the fourth ventricle early and permanently, and cerebral decompression will not give relief. A tumor a little distance from the third ventricle, in the





ILLUSTRATING DOCTOR HOLDEN'S ARTICLE, "AN INTRODUCTION TO NEUROLOGICAL OPHTHALMOLOGY."

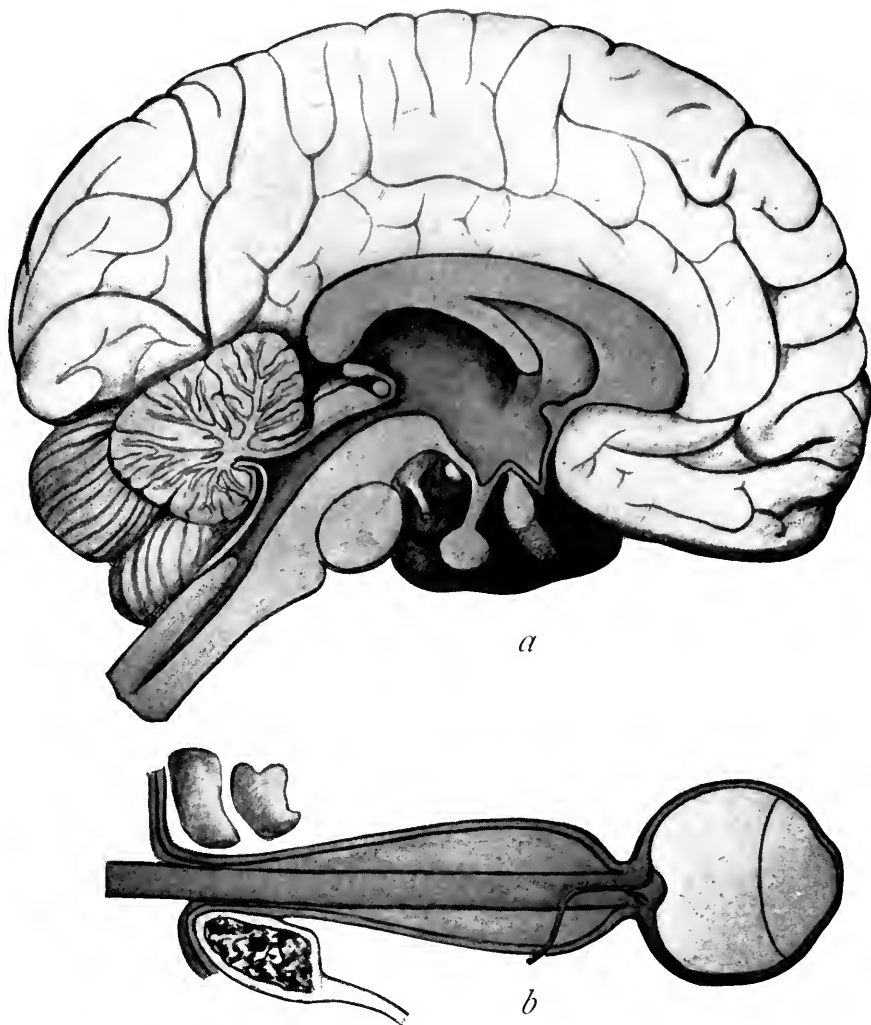


FIG. 3—(a) A diagram showing the third and fourth ventricles and the aqueduct of Sylvius. (b) Distention of the intervaginal space of the optic nerve with cerebrospinal fluid in papilledema.

cerebellum, for example, will block the passage by compression fairly early. In such cases cerebellar decompression gives relief. Tumors distant from the third ventricle, in the frontal lobes or in the pons, for example, do not block the passage until late in their course and decompression may not be indicated. (See Fig. 3.)

The liquid in the subdural space of the brain, when forced under pressure into the subdural space of the optic-nerve sheath, distends this space, compresses the central artery and vein of the retina which pass through it, and to some extent strangulates the nerve head. The nerve head then becomes edematous together with the retina about it. The retinal arteries are narrowed and the veins are dilated and tortuous, from compression. After some months the nerve fibers are compressed and atrophy of the optic nerve ensues.

With the ophthalmoscope we find in the beginning a slight edema of the nerve head which veils its nasal margin. Then the upper and lower margins of the nerve head become veiled, the veins are dilated and tortuous, the nerve head and surrounding retina become swollen with edema, and often hemorrhages appear in the retina. Some weeks after the onset the swelling of the retina subsides to some extent and the nerve head as a rounded, grayish, pulpy-looking mass projects one or two millimeters above its normal level. This fourth stage of papilledema may continue for months. Later, a general pallor of the nerve head appears through the edema, as atrophy of the nerve sets in. The swelling of the nerve head from this time subsides gradually. As papilledema does not develop in a nerve which is already atrophic, so the papilledema subsides when the nerve becomes atrophied. For months or years longer the pale nerve head has blurred margins and the retinal veins remain dilated and tortuous.

Visual disturbances are noticed early. The patient complains of a cloud over his sight which annoys him. When his central vision is tested with a well illuminated test card it will be found to be normal or nearly so, but examination of the visual fields will show them to be defective. There may be or may not be concentric contraction of the field for white, but concentric contraction of the fields for colors is the rule. In a few cases pressure upon the chiasm, the optic

tracts, or the optic radiations will add symmetrical hemianopic defects to the existing concentric contraction of the fields.

After a time central vision deteriorates. Fluctuations of vision then occur. On days when the intracranial pressure is particularly high the headache will be more severe and the sight will be more blurred. Vision in one or usually both eyes may be lost completely for a few seconds or minutes at a time and such attacks may occur at very frequent intervals. When this stage is reached decompression cannot be longer postponed. The reduction of pressure by the operation dispels the cloud before the sight and leads to enlargement of the contracted visual fields. If, however, the vision has been reduced through atrophy of the optic nerve, further atrophy may be checked by the operation, but the lost vision will not be regained.

The cerebral decompression operation for the relief of failing vision is commonly regarded as being a procedure of very recent introduction, but, as we are learning more and more that our new ideas were already known to the ancients, it does not surprise us to find in the Hippocratic Writings (Hippocrates lived 2400 years ago) a passage on decompression which runs roughly as follows: "If vision fails without demonstrable disease of the eyes, incise the scalp, separate the soft parts, and remove a portion of the bone of the skull, thus releasing the accumulated liquid that by its pressure has caused the deterioration of vision."

Intracranial hemorrhage may cause increased pressure with a certain degree of papilledema when the blood pressure rises as shock passes off. This papilledema is a symptom of value in confirming the existence of increased intracranial pressure, but in itself does not cause disturbances of vision or require cerebral decompression for its relief.

#### OTHER ANOMALIES

*Retraction of one or both upper lids* is a manifestation of excitation of the cervical sympathetic nerve, which causes a contraction of the small unstriped muscle in the upper lid supplied by the sympathetic. This is sometimes an early sign of exophthalmic goiter. It is this retraction of the

upper lid that gives rise to the Graefe symptom in exophthalmic goiter—the lagging behind of the upper lid when the eyes are slowly turned downward.

*Pulsating exophthalmus* is usually due to a fracture in the middle fossa of the cranium which tears the internal carotid and permits communication between the carotid artery and the cavernous sinus. Arterial blood is forced back into the veins of the orbit and causes protrusion of the eyeball. The diagnosis of this arterio-venous aneurysm is verified by the bruit heard with the stethoscope when placed on the temple or by the subjective pulsation and bruit experienced by the patient.

**The psycho-genetic disturbances of the eye:** In neurasthenia, psychesthesia, hysteria, the traumatic neuroses, the different varieties of shell-shock, and in simulation, ocular disturbances are found, which may be classified as anesthetics, spasms of the voluntary muscles, and disturbances of vision. The anesthetics or hypesthesias occur in one eye or in both and are revealed by touching the cornea with a piece of cotton, when the normal reaction is found to be absent or diminished.

The spasms comprise (1) a tonic or clonic contraction of the sphincter oculi, causing a closure of the lids, or when of lesser degree a drooping of the upper lid (ptosis), which is often found to be spasmodic and not paralytic, since the patient resists attempts to raise the lid by force; and (2) a spasm of the internal recti, by which the patient produces a convergence of the eyes and sees double. If he says that he sees with one eye a light single at one distance and double at another distance, the disturbance is unquestionably functional.

The visual disturbances are subjective or objective. In the former the patient sees flashes of light or spots and clouds before his vision, or objects appear larger or smaller than they should, due to spasm of accommodation, or images usually suppressed are recognized, so that, for example, the patient may complain of seeing his nose, or after-images may be of long duration, or a host of other purely subjective disturbances may be complained of.

Besides these there are disturbances of vision capable of demonstration. First, there is concentric contraction of the field for small white and colored test-objects with normal

acuteness of central vision. In easily fatigued persons this contraction is progressive as the test proceeds and forms a diminishing spiral. Second, with some concentric contraction there is reduction of central vision and the letters on the test card are not read so far down as the patient's refractive condition should allow, but by forceful urging many of these patients can be induced to read the test letters as far down as they should. Third, there may be a fixed diminution of central vision or a fixed defect of almost any character in the field of one or both eyes. These defects are likely to be suggested by the first examiner and to be fixed in the patient's mind at that time. Fourth, there may be blindness more or less complete in one eye, which can be unmasked by some of the tests used by the oculist to detect simulated blindness. Fifth, there may be apparently complete blindness in both eyes. This condition is usually an asset to the patient and a measure of defense against something he wishes to avoid seeing or against some task from which blindness relieves him. In all these cases the optic discs are normal in appearance and the pupils are active or but slightly sluggish.

The functional nature of the condition is betrayed, as a rule, by the lack of co-operation of the patient in moving his eyes. If directed to turn the eyes to the right, for example, he apparently makes a great effort to turn the eyes in that direction, but in reality makes an effort not to turn the eyes to the right. The presence of hypesthesia of the cornea and other functional disturbances supports the diagnosis, and his recent psychic experiences, which can be learned from his associates, together with his obvious desires, confirm the diagnosis.

## A CASE OF LEUCOSARCOMA OF THE IRIS, TREATED BY RADIUM.

BY DR. SANFORD GIFFORD, OMAHA, NEBRASKA,  
1ST LIEUTENANT, M. R. C.

*(With two illustrations on Text-Plate XIV.)*

**S**ARCOMA of the iris is not so rare that the report of such a case alone would be particularly valuable, but the opportunity for pathological examination after systematic treatment by radium is so unusual as to be worth recording.

CASE.—The patient was a man of fifty-two years, who was referred to Dr. Harold Gifford on February 10, 1915. He reported that for two or three years he had noticed a small spot on the lower part of his right iris, which had been gradually increasing in size. No subjective symptoms. Present condition: L. E. normal vision,  $\frac{20}{20}$ ; R. E. showed a pinkish, somewhat lobulated tumor about  $\frac{3}{16}$  of an inch in diameter, taking in the greater part of the lower and inner quadrant of the iris, projecting forward so as to touch the cornea, leaving but one millimeter of the iris root apparently free. In other respects normal. Vision  $\frac{20}{80}$ . (See Fig. 1.)

The difficulties and dangers of the situation were explained to the patient and he decided to try treatment with radium, rather than to have the eye enucleated or the tumor removed. From February 11th to the 20th, he received a treatment daily with from 32 to 48 milligrams of radium bromide, each treatment lasting from twenty to thirty-five minutes, the tube being applied to the outer surface of the closed lids in all of the treatments except two. At these two treatments, the tube was held directly across the cornea by conjunctival sutures. The immediate result of this treatment was that about February 25th, after he had returned home, he developed a right-sided facial paralysis which Dr. Quigley has informed me he has noticed in other

cases where radium has been used freely. This paralysis persisted for nearly a year and then gradually disappeared. The tumor diminished somewhat in size after the lapse of a month, but by the middle of May it again showed slight signs of increasing, and Dr. Quigley applied the radium in the same amount as before for nearly two hours with the tube directly in contact with the cornea.

He was not seen again until September 11, 1917, when he returned stating that the eye had given him quite a little trouble with pain and irritation for several months, and as his home physician had advised him to have the eye enucleated, he came down to have it done. The tumor was found to be decidedly smaller than when he was first seen, the portions in the chamber proper having largely disappeared. But it had become adherent to the cornea which showed a vascular pinkish area about an eighth of an inch in diameter, extending through the entire thickness and slightly raising the surface. From the posterior surface of the cornea, the tumor ran down and back to a small attachment at the extreme periphery of the iris. The eye was moderately congested and irritable, and the fundus could not well be seen on account of the opacities in the lens capsule and in the vitreous. The vision was  $\frac{2}{20}$ , not improved by glasses, and the tension raised to forty-five millimeters as opposed to twenty-three in the other eye. In other words, the condition of the tumor itself was relatively favorable, but the eye seemed to have suffered so much from the effects of the radium and the patient was so determined to get rid of it that it was enucleated. Unfortunately, no photograph of the patient was taken before enucleation, which would have showed the marked change from the condition seen in Fig. 1.

#### PATHOLOGICAL REPORT.

*Iris:* At the pupillary margin, the iris is thickened, forming a club-shaped prominence forwards. This is raised about 30 microns from the anterior surface of the iris. It is composed of large spindle cells with dark staining oval nuclei, arranged in whorls around dark staining round oval bodies of various sizes which seemed to be the nuclei of the spindle cells in cross-section, though some may be round cells. No mitoses are found. The spindle cells merge into the stroma, getting smaller, and in the stroma no round cells or sections of large nuclei are found. It appears normal except for one place at the root, where it is adherent to the cornea, and for one area of lymphoid infiltration. No pigment is found in the tumor part, and the arrangement of



ILLUSTRATING DR. GIFFORD'S ARTICLE ON "A CASE OF LEUCOSARCOMA  
OF THE IRIS, TREATED BY RADIUM."



FIG. 1.

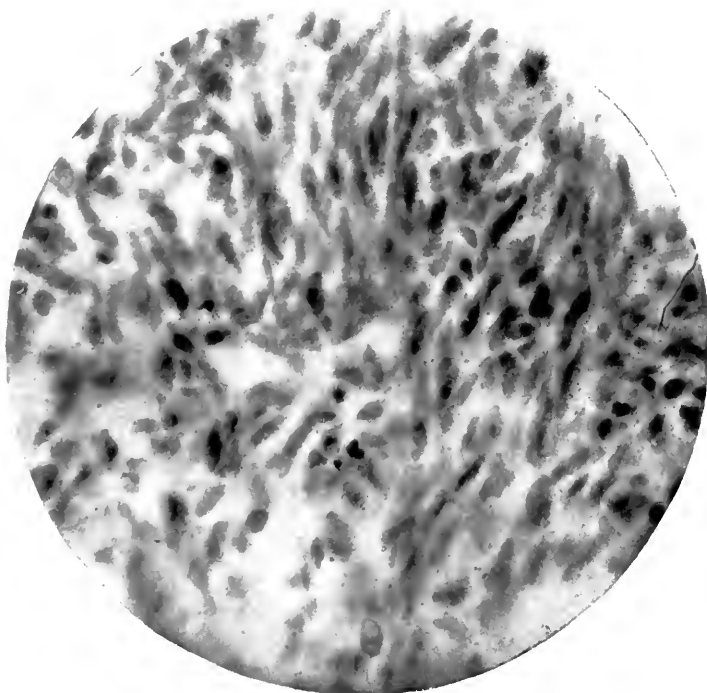


FIG. 2.



pigment in the rest of the iris is normal. At the root where it is adherent to the cornea, there is slight infiltration with polymorphs and leucocytes in its anterior layers. The above changes are found only in the sections of about 2 millimeters of the lower central iris. (See Fig. 2.)

*Cornea:* The endothelium at one place near the iris angle is adherent to the iris, and is wrinkled and defective. The corneal layers over this are infiltrated with polymorphs, lymphocytes, and connective tissue cells running in irregular directions. Under the epithelium are three large vessels in section surrounded by polymorphs and round cells.

*Ciliary Body:* In the above sections, there are a few round cells at the base of the ciliary processes. In sections taken near this, but not showing the tumor, is seen a collection of cells with dark staining round nuclei. The round cells spread out into the ciliary processes and back into the muscle. A few pigment cells are seen among them. (In the iris, about halfway to the pupillary border, next the pigment epithelium, is a small group of these round cells.) These cells are smaller than the cells of the tumor, regular in size and shape, and appear like lymphocytes.

**ANATOMICAL DIAGNOSIS:** Spindle-celled sarcoma of the iris with secondary reparatory changes.

Subacute and chronic inflammation of cornea at one point with adhesions to iris.

Lymphoid infiltration of ciliary body and root of iris.

Levin and Joseph (1) have recently described conditions found after treatment by radium of cancers of the breast and other regions, which they call sterilization of the neoplastic cells. By this they mean that the cells are still present, unchanged or in various stages of regression, and often surrounded by connective tissue, while the tumor has not increased in size, nor the cells spread to other regions.

Morson (2), Wassermann (3), and Prime (4) reached the same conclusion, the last two after treating mouse cancer cells in vitro by radium and finding that, while remaining alive, they cease to show mitoses and fail to produce cancer when injected into healthy mice.

Levin and Joseph suggest that this loss of reproductive power is the first stage in a cure of cancer which is followed by the ageing and death of the cells. They believe this is the condition in cases of clinically cured cancer when the tumor remains, but does not increase in size or form mitoses, for a

sufficient length of time to preclude the chance of a spontaneous recurrence.

The case here reported seems to present much the condition which they describe, the cells showing no mitoses or evidence of spreading into neighboring tissues. About two hundred sections were cut out and specimens examined at frequent intervals from the whole circumference of the globe, so it is certain that there were no other foci. The primary growth has been reduced in size.

DISCUSSION: The spindle cells seem to be the characteristic cells of the tumor. The round cells in the ciliary body and the small collection in the iris are different from the cells of the growth, and would seem to represent a lymphocytic reaction such as usually follows treatment by radium.

The adhesion to the cornea and the reaction around it are probably the result of direct contact of the tube of radium with the cornea. There is no evidence of extension of the tumor at this point nor is the pink area in the cornea part of the tumor, as implied in the clinical record.

The loss of vision may be due to vitreous opacities formed in reaction to the radium or to organization of exudate formed when the tumor was growing.

THERAPEUTIC DEDUCTIONS: The two accepted treatments for sarcoma of the iris are iridectomy and enucleation. Wintersteiner (5) well sums up the opinion on these two measures. The commonest opinion is in favor of primary enucleation except under certain well defined favorable conditions.

For Wintersteiner these are met only when:

1. Vision in the eye is good.
2. The iridectomy offers no obstacle to complete removal.
3. The tumor is small, slow-growing, well defined, and at the pupillary edge. It must not reach into the anterior chamber, touch the lens or cornea. There must be no other pigment flecks or diffuse staining of the iris. Tension must not be increased. Under these conditions he would do iridectomy followed up by observation and enucleation at first sign of recurrence.

On the other hand, he says, Kerschbaumer, Wood, Pusey, Coffey, Vaucleroy, and Schleich always advise primary enucleation.

Wintersteiner points out that there is some danger of iridectomy's producing metastasis but this is also true of enucleation, and cites six cases of metastasis after primary enucleation.

I can find no other record of the use of radium in new growths of the iris. It has been used with some success, however, in other intraocular tumors.

Deutschmann (6) reports treating by mesothorium a melanoma of the choroid in the left eye of a man of 51 whose right eye had previously been enucleated after an accident. .01gm of mesothorium in a capsule was sewed into a conjunctival pocket and left for one to two hours. Nine days after this the tumor could be seen to be flattened. This was repeated three times at one-month intervals and then four times at two-week intervals. The subretinal effusion was withdrawn three times by puncture. At the end of this five-months period the marked projection first seen in the fundus was replaced by a series of folds and vision was fingers at three inches, when the case was lost sight of.

C. F. Clarke (7) treated sarcoma of the orbit by inserting a tube of radium in the orbit after canthotomy and division of the external rectus. It was left in four hours and this was repeated every two weeks. The proptosis quickly receded and in two years the case was apparently cured. It is interesting to note that a nuclear cataract developed after six months of the treatment, perhaps due to the radium.

Others have reported good results in treatment of recurrences in the orbit after enucleation for sarcoma (Sattler) and neuro-epithelioma (Theobald).

The most careful work was done by Axenfeld. He reported in 1915 (8) results eighteen months after treatment by radium and mesothorium of a case of glioma in a child whose right eye had been removed for glioma. At operation three foci of growth, one rather large, were seen in the left eye, and radiotherapy was begun. He made eleven careful fundus examinations, noting the decrease of the growth, and eighteen months after the first treatment reported that one area had disappeared, another was almost gone, and the third, the largest one, had very much decreased in size. In 1916 (9) he reports further progress: "The largest growths had completely dis-

appeared, leaving only an evidently dead preretinal trace," with no recurrence elsewhere.

He quotes Hillgärtner, who used the X-ray in bilateral glioma, and after 84 treatments reduced a tumor that filled the vitreous to  $\frac{2}{3}$  its former size. In the other eye the beginning of a glioma, which at first could be plainly seen, was completely resorbed. He says that Würdemann by X-rays stopped a glioma which had invaded the orbit and sinuses, and which had recurred repeatedly after simple exenteration. Also that Pusey was successful with X-rays in preventing recurrences of glioma and sarcoma after enucleation.

Axenfeld takes up carefully the question of harm to the function of the eye done by radiotherapy, which would, of course, occur to anyone who thought of using such treatment.

The experiments of Birsch-Hirschfeld showed a distinctly harmful effect on the eye, especially the retina of animals exposed to X-rays. Rauch's results were similar. Axenfeld points out, however, that they used unfiltered rays in their experiments, and that his own experiments, carried out with filtered rays, showed no demonstrable damage to the eyes. He used aluminum filters for his radium 3-4mm thick which excluded all but the hard, deeply penetrating gamma rays. For mesothorium, which he used twice, he used a lead filter. His course was given in four groups of treatments of 6, 6, 3, and 2 treatments each in which doses of 10 to 35 of radium were given for 8 to 32 minutes at two- to ten-day intervals in each group and a month between the groups (two months before the last group).

As to the damage done to his case, nothing could be certain till it became old enough for subjective tests.

In the case here reported, some of the changes in the globe were almost surely the result of the rather intensive radiation. Such were the vitreous opacities and a slight diffuse cloudiness of the cornea. No changes were observed in the retina which was examined, though loss of part of the specimen in cutting prevented a very careful examination of the whole retina. The opacities, however, were probably sufficient to account for his loss of vision from  $\frac{2}{3}$  to  $\frac{2}{10}$ . The filter used was silver.

In considering the possibility of treating by radium or X-

ray it is apparent that the iris offers very favorable conditions for such treatment. The rays reach the growth through the clear cornea almost as if it were on a skin surface, and the results of the treatment are as open to observation. If it is unsuccessful and the growth continues to increase, operation may be resorted to with about as good a chance of success as if it had been done at first. It may be with even a better chance, as even partial sterilization of the tumor cells would lessen the danger of their multiplying if some of them should be forced into the circulation by the operation.

Though the results from such treatment in intraocular tumors have been so far rather meager, some of them seem very positive. It would seem, then, especially in tumors of the iris, that treatment by radium should be seriously considered. Where the growth is not especially rapid, even if it has reached the iris root, such treatment should be offered to the patient as an alternative to operation, he being observed carefully during each treatment. Where operation is refused, it would seem only just to the patient to advise radium very urgently.

I take occasion here to extend my hearty thanks to Dr. D. T. Quigley, for accurate records of treatments, to Dr. Chester E. Waters for examining the sections, and, most of all, to Dr. Harold Gifford for permission to use the material and assistance in reporting on it.

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## SOME COMMENTS UPON THE PROBABLE CAUSE OF EXOPHTHALMIC GOITER.

BY DR. JOHN DUNN, RICHMOND, VIRGINIA.

IN the *Archiv für Augenheilkunde*, lxxvii. Band, Heft 1, 1914, Prof. Dr. Schoenborn, in an article on the "Pathology and Treatment of the Diseases of Internal Secretion," says of Basedow's disease that the primary cause of its production is in the great majority of cases obscure. It has been attributed to "both psychic and somatic insults"; further to infectious diseases (sore throat, typhus, scarlet fever, syphilis, and tuberculosis), to primary infection, to tumor of the gland itself, and finally to drug poisoning (iodine), and to overuse of the extracts of the gland itself and, perhaps, to intestinal intoxication. Prof. Schoenborn continues: "The uncertainty which surrounds its genesis and etiology makes itself felt in the treatment." Then follow comments upon the better known therapeutic and surgical suggestions, the interesting feature about which suggestions is that *not one of them takes into consideration the possibility of a therapy which is based upon the removal of an active cause*, and thus obtaining a cure. For the views which are expressed in this article no originality is claimed by the author. They are an outcome of the practical application of the results of the work done by others in tonsillar pathology; in especial the studies in focal infection by Rosenow and Billings. The excuse for reporting the following cases is to be found in the fact that Basedow's disease has problems which may not be excluded from the domain of ophthalmology. The various phases of the ocular picture in ophthalmic goiter are well known to all oculists, and any suggestions which offer a probable definite, constant, contributing, if not also originating, cause of hyperthyroidism



are worthy of consideration by them. The cases selected in this paper for illustration seem to prove that in acute thyroiditis (Case 1), and in hyperthyroidism (Cases 2, 3, and 4) *the main agent at work in causing the continuance of the thyroid changes is an infectious one.* From this standpoint our problem in any given case resolves itself into discovering the source of this infection and a discussion of the possibility of its removal. The question as to what it is in the first place determines that the infectious agent in the blood should select the thyroid substance rather than a joint or the endocardium for its focal action, probably finds its answer in the following: Trauma, inherited lack of resistance, and functional exhaustion. Granted that the conditions in the thyroid have been rendered favorable for the reception of certain infectious agents in the blood and that the gland becomes inflamed, then there develops within the thyroid a selective affinity for those same varieties of microorganisms whenever thereafter they are in the blood in excess of the blood's ability to dispose of them. This same thing applies to other parts of the body and explains the recurrence in iritis, focal choroiditis, endocarditis, articular rheumatism, etc. Our problem in treatment, other than surgical, of the thyroid then resolves itself into two parts. The one is to free the body from the presence of the foci which contain the special microorganism. The other is to strengthen the resisting powers of the body. That the latter, which has heretofore consisted chiefly in prolonged rest and selected feeding, has at times been followed by a marked lessening of the hyperthyroidism is well known. The aims of this form of treatment are, however, too indefinite to be used except as an adjunct or as a *dernier ressort*. That the importance of the search for a chronic focus of infection in the treatment of Basedow's disease is not generally recognized is shown by the fact that surgical effort, save in a few places, is almost exclusively directed towards the overacting thyroid, the cause of the symptoms of hyperthyroidism, rather than towards a chronic focus of infection as the cause of the thyroid changes.

CASE I.—Mrs. I., aged 26, had an infection of the left lower lid which resulted on the third day in an abscess with considerable swelling of the adjacent structures. The patient's neck began to swell and when I saw her on the fourth day

there was a marked enlargement of the thyroid with equally marked symptoms of acute oversecretion on the part of the gland. "Doctor, isn't it strange that I should have both the lid trouble and the throat trouble at the same time?" the patient asked me. The lid abscess was opened and a solution of nitrate of silver was applied to the cavity. A mercurial was ordered and in less than a week the thyroid enlargement and the symptoms of hyperthyroidism had disappeared. At the time this case was under my care, I had not associated thyroid disturbances with tonsillar disease and the tonsils were not examined. The case, however, was in my experience so unusual that I made notes of it. Whether or not the thyroid inflammation was secondary to infection from the lid abscess or whether both were to be referred to a focus of infection selected elsewhere in the body, I cannot positively state. The prompt disappearance, however, of the thyroid disturbance following the treatment of the lid abscess made me at the time believe the former to be the case. If so, it is difficult to understand why more cases of a similar nature have not been reported by ophthalmologists.

CASE 2.—The second case is that of Mrs. L., aged 36, and belongs to the type of thyroid disturbance which may be termed subacute hyperthyroidism. The thyroid was much enlarged and soft. There was a moderate exophthalmos, remittent attacks of increased pulse rate, and an almost continuous mild degree of abnormal nervousness, the details of which Mrs. L. could not put into words. Both tonsils had chronically diseased crypts. The enucleation of the tonsils was followed, without other treatment, by so marked a reduction in the size of the thyroid as to render its hypertrophy demonstrable only by palpation and by the disappearance of all the symptoms due to hypersecretion on the part of the gland and by recession of the eyeballs. This case has several points of interest. Some years before Mrs. L. had been treated by me for an attack of acute hyperthyroidism. I had at the time not suspected the possibility of a chronic focus of infection. The acute symptoms subsequent to (rather than as the result of as I now think) a course of general (!) treatment subsided, leaving a somewhat enlarged thyroid with a subsequent history of dysthyroidism. The tendency of this case was more and more towards the development of continuous hyperthyroidism and I believe at the time the tonsils were removed that a full-fledged acute case of Basedow's disease with excessive exophthalmos was imminent. The prompt disappearance of all symptoms of thyroid disturbance can be attributed only to the removal of the tonsils, or rather, to

the removal of the chronic foci of infection which the tonsils contained. As I now see the original attack of acute thyroiditis, I believe it was secondary to tonsillar infection.

CASE 3.—A further example of this type of thyroid disturbance is the case of Mrs. L., aged 50. The occasion of her visit was the sensation as though her collar were too tight and this although her neck was bare in front. At times this sensation was so pronounced as to cause her to feel as though she were choking. Examination revealed a deep-seated enlarged thyroid. The central lobe seemed not to participate in the hypertrophy. There was the accompanying history of mild, though pronounced, hyperthyroidism. The point of interest for us here is that the enlargement in the neck "came on at the time *I had an abscess in my throat four years ago.*" Neither tonsil was healthy. In this connection two questions will probably be asked. What constitutes a chronically diseased tonsil? And why, so long as the tonsil remains in situ and diseased, should there be any subsidence of the symptoms? The former question has been rather fully gone into in an article entitled "The Tonsil as a Source of Infection in Iritis" (in ARCH. OF OPHTHALMOLOGY, vol. xlv., No. 5, 1917). Without entering at length into the second question, it may be stated that the entrance into the blood of infectious agents from a diseased tonsillar crypt is not necessarily at all times constant. The abrasions of the mucous areas of the crypts caused by the presence of microorganisms, their products and cast-off epithelial cells may at times heal over and remain healed for a time, and this is rendered possible not only by the protective shutting off of the region of the abrasions such as takes place about all chronically ulcerated places, but further by the fact that from time to time the emptying out of the contents of the crypts does away for the time being with the element of pressure against the walls of the crypts. Fresh ulcerations recur with a sufficient accumulation of the above-mentioned substances. There is, besides, a further reason: the quantity of microorganisms entering the blood from these foci of infection even when constant may be less than that which can be destroyed by the blood cells and so none reach the region of selective affinity (in this case, the thyroid). Thus we can understand how anything which lessens the "resisting qualities of the blood" increases the probability of disturbance in any given area of "selective affinity."

CASE 4 is that of Mr. G., aged 28, and is one of Basedow's disease, of between three and four years' standing; at time of his visit in its acute stages. Both eyeballs showed a high degree of exophthalmos with marked Graefe's sign. A

large bulging thyroid extended entirely across the front of the neck. Patient was unable to sleep, unable to digest his food, unable to do any work, or to use his eyes for reading or writing. His general nervousness was pitiable to behold. Pulse rate was high. Both tonsils were diseased. Double tonsillectomy was done. Three months later there had taken place such a change in Mr. G.'s appearance that I failed to recognize him when he called at my office. The exophthalmos had entirely disappeared. The thyroid had so diminished in size as not to be noticeable on careful inspection. Mr. G. stated he now slept all night and could eat what he wanted to and work as much as he pleased. His nervousness had disappeared. He said, "I am well." There, however, remained an easily excitable pulse and on palpation the thyroid was still enlarged. Two months later the same condition of the pulse persisted and there was a slight, although demonstrable, further enlargement of the thyroid. Mr. G., however, was according to his own accounts "well." I do not, however, consider him so. The tendency for the thyroid to enlarge indicates that there persists somewhere in his body an additional focus of infection which is still at times pouring into the blood microorganisms in a quantity sufficient to reach the weakened thyroid. As yet I have been unable to decide where this focus is. This case is not reported as a case of cured Basedow's disease, but because it serves to accentuate the probability that hyperthyroidism is the result of infection and that at times the focus may be situated in the tonsillar crypts. The interesting feature of this case is the entire disappearance of the exophthalmos, a symptom which often persists for a long time after surgical operation upon the thyroid. Taken together these four cases seem to show that infection within the tonsil is one of the causes of enlargement of the thyroid. Since my attention was directed to this subject, I have examined the throat in all cases of Basedow's disease which have come to my office and in all, save two of them, there was demonstrable disease of the tonsils, and in no one of them had a connection between the diseased tonsil and the hyperthyroidism been suspected. There may have been disease of the tonsil in these two. One was seen before I appreciated the importance of chronic tonsillar disease in its relation to inflammatory troubles in distant organs. In this case I did not examine carefully the crypts of what was an apparently normal organ.

I have examined also the throats in a considerable number of cases of simple goiter occurring in girls and young women, and with few exceptions all of them had demonstrable disease

of the tonsils. The coëxistence of tonsillar disease and hypertrophy of thyroid does not necessarily prove that the latter is the result of the former, but taken in connection with the results obtained in such cases as the above, and I have had a considerable number of similar results, we may safely state that chronic tonsillar disease (and the same would apply to any other chronic focus of infection anywhere in the body) may at least determine exacerbations and continuance of the thyroid disturbances. In regard to simple goiter, it is significant that the vast majority of them begin in youth when the tonsils are most subject to acute inflammation, and in young women at a time when the normal functioning of the thyroid can be most easily rendered unstable through its interrelationship in its activities with the ovaries.

The subject of simple goiter is foreign to the purpose of the paper and further comment is omitted.

In this connection it will be of interest to comment upon three cases seen lately of Basedow's disease which had been subjected to thyroidectomy. Mrs. M., aged 36, had one half of the thyroid removed. Four months later the exophthalmos which had been excessive at the time of the operation showed no recession. There was no diminution of the pulse rate or lessening of the other nervous symptoms. *Both tonsils were enlarged and inflamed.* Seven months later—that is eleven months after the operation—the thyroid hypertrophy had disappeared. The exophthalmos had greatly lessened, but much more in the left than in the right side. The patient was still extremely nervous and pulse rate high. Her sleeplessness had in a great measure disappeared. Her appetite had returned, and her ability to take exercise had returned. It is of interest to note that within these last seven months the active tonsillar inflammation had subsided. In this case it is probable that there will be a recurrence of the thyroid disturbance should there be further active tonsillar inflammation.

In the case of Mrs. G., aged 39, operated upon eleven years previously, there had never been any exophthalmos. The ocular symptoms consisted, according to the patient, of a tendency of the pupils to dilate abnormally. This has disappeared, as have the nervous symptoms from which Mrs. G. suffered. Both tonsils show hypertrophic changes and there

is a history of many attacks of sore throat. This case, which may be considered cured, was probably of a mild type and it is probable that almost all of the thyroid was removed.

The third case is that of Miss L., aged 24. A part of the thyroid was removed in 1912. In March, 1917, the symptoms being worse than they were prior to the first operation, a further resection of the thyroid was done. Six months later the nervous symptoms had disappeared. There was never marked exophthalmos. There is, however, still present the exophthalmic stare without any protrusion of the eyeballs. This apparent lack of normal mobility of the eyes told the story of Basedow's disease, before the neck was looked at or any questions asked. In this case, the appearance of the neck suggested that only a small portion of the thyroid was left after the second operation. *Both tonsils tell the story of repeated infections.*

*Conclusions.* When for reasons either inherited, functional, or traumatic, the normal resistance of the thyroid has been so lowered that it becomes a "receptive focus" for any special species of microorganisms, there may in the future result thyroid disturbances of varying degrees of intensity whenever these same microorganisms are present in the blood in numbers greater than the blood's ability to digest them. A *chronic* focus of infection is the most common source of these microorganisms. While, in any given case, the focus may be situated anywhere in the body, *the most common location in the case of both simple thyroid hypertrophy and hyperthyroidism is the tonsillar crypts.* Where the focus can be entirely eradicated, the overactivity of the thyroid can be done away with. How far recession in the size of the thyroid is to be expected after the removal of the focus will depend upon the amount of fibrous changes already present in the gland. That the removal of a chronic focus of infection will do away with the special receptivity previously developed within the thyroid is not to be expected and recurrence of inflammatory activity may take place whenever in the future a new focus of infection containing the especial microorganisms is established. This, together with the further fact that there may coexist two or more foci of infection, should be borne in mind when considering the prognosis in a case of Basedow's disease.

The difference between the subacute and acute type of Basedow's disease is only one of degree, representing different resisting powers on the part of the thyroid. In some cases, the microörganic invasion of an injured or functionally exhausted thyroid may result at once in a pronounced type of Basedow's disease. In other cases, the invasion results in changes in the thyroid which bring about exacerbations of various degrees of severity, and this type of dysthyroidism in the presence of a distant focus of infection may persist for years. No immediate disappearance of the hyperthyroidism follows the removal of the focus of infection. Usually three or four months have to elapse before the full effects of the operation are to be seen.

## THE RETURN OF TENSION AND THE OCCURRENCE OF RETROCHOROIDAL HEMORRHAGE AFTER TREPHINING.<sup>1</sup>

BY DR. ARNOLD KNAPP, NEW YORK.

THE two conditions illustrated by the following cases, namely, the return of tension and the occurrence of a retrochoroidal hemorrhage, are in no way to be ascribed to the trephining operation *per se*, as they have been observed after any form of operation devised to reduce abnormal intraocular tension; and in the writer's opinion both of the accidents described in the following cases would have occurred in even a more aggravated form after an operation where a large incision of the sclera was required. The above title is therefore perhaps misleading.

A case illustrating the non-reduction of tension after operation is the following:

L. H., aged 59, had lost one eye from glaucoma, and in her remaining eye vision was reduced to movements of hand in the temporal field with a tension of 42. Trephining was done in the usual manner on June 26, 1917. A  $1\frac{1}{2}$  mm disk was removed, and a large piece of iris excised. At the completion of the operation the tension of the eye seemed hard to the finger. On the following day this hardness was still present. On the second day after operation, on examining the area of the trephine-opening, a circumscribed bulging was found present. Tension measured 60 with the tonometer. A posterior sclerotomy was then performed. This was followed by a reduction of the tension and the bulging at the trephine-opening was flattened, and on July 7th

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<sup>1</sup> Read before the Section on Ophthalmology, New York Academy of Medicine, January 21, 1918.



the tension registered 26. There was perception of light and on oblique illumination blood could be seen in the vitreous. On July 10th the tension had risen to 60 and the trephine-opening was again bulging. The anterior chamber was of medium but of uniform depth, so that there seemed to be no indication of a displacement of the lens. On July 11th it was decided to investigate the trephine-opening, and the conjunctival flap was again reflected. The opening in the sclera was found to be filled with a large bead of vitreous. This ruptured and a considerable quantity of fluid vitreous escaped. The flap was replaced and sutured. The healing after this was uneventful and the tension of the eye remained down until July 21st, and two days later the area of trephine-opening was again bulging and the tension was 80. The iris level was uniform, the lens was clear, no details could be seen in the fundus. On the following day another posterior sclerotomy was performed which lowered the tension; and on July 26th, the tension still being down, another trephining was practiced on the temporal side of the old wound without any accident. There was no reaction following this last operation, and apparently the tension has remained down. On August 4th the patient returned to her home in New York State with a tension of 20, perception of light, and blood still visible in the vitreous. A letter received on February 11, 1918; states that the eye has not given her any further trouble and that she can count her fingers.

Operation in certain exceptional and generally acute congestive glaucomas has been followed by an aggravation of the glaucomatous symptoms. This condition was recognized by von Graefe and designated "malignant glaucoma." It has been variously explained, generally by prolapse or traction on the ciliary body. Friedenwald<sup>1</sup> reported on a case of malignant glaucoma and collected the histories of twenty-four cases. In these cases, shortly after an iridectomy there was increase in tension with obliteration of the anterior chamber and all the symptoms of an intense congestive glaucoma together with a rapid loss of light.

Return of tension at some time after operation in chronic glaucoma has been observed in a definite proportion of cases irrespective of the operative procedure, and is generally due to the closing, or the lack of the proper establishment, of a

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<sup>1</sup> Friedenwald, ARCHIVES OF OPHTHALMOLOGY, 1896.

filtrating scar. The persistence of tension in cases of chronic glaucoma after trephining is due, according to Elliot,<sup>1</sup> to either a dislocated lens or prolapse of vitreous, provided that no uveal tissue obstructs the trephine-opening. The usual cause for these complications is a vitreous hemorrhage which, while it may be facilitated by certain errors in the operative technic, is often beyond the control of the operator. Contributing causes are a relaxed suspensory ligament of the lens from an anteriorly displaced lenticular apparatus and degenerative changes in the vitreous body.

The dislocation of the lens shows itself by a change in the depth of the anterior chamber. This complication, which the writer has fortunately not encountered, is a very difficult one to deal with. Prolapse of vitreous is a more frequent complication and can be readily detected by a bulging at the trephine-opening, the anterior chamber remaining of uniform depth. In the cases of this condition which the writer has observed the tension did not go down and it was necessary to reflect the flap and incise the vitreous hernia. This was followed by the escape of a small quantity of vitreous; the healing, however, was uneventful, and the result satisfactory, except in the above-mentioned case where increase of tension returned and posterior sclerotomy and another trephining became necessary.

The following case is one of detachment and dragging of the retina into the trephine-opening, presumably due to retrochoroidal hemorrhage following a trephining for glaucoma.

The patient was a girl, aged 13, who presented the unfavorable combination of chronic glaucoma and subluxated lenses in both eyes, and stated that her sight had been poor for five years. The subluxated lenses were either due to the glaucoma or a coincident condition, surely not a cause for the glaucoma. With her myopic glasses ( $-7$  D) the vision was R 20/200, L. 20/100. The pupils were irregular and eccentric. The depth of the anterior chamber was not uniform and there was a definite tremulousness of the iris. The anterior sclera in both eyes was unusually blue, suggesting a distension of this part of the eyeball. The tension with tonometer in both eyes measured from 60 to 70°. The fields showed a concentric contraction to

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<sup>1</sup> Elliot, *Sclerocorneal Trephining*, II. ed., p. 111.

about 10°. Both optic nerves were cupped and atrophic. A brother of the patient was also nearsighted and the ocular tension varied from 38 to 40; the optic nerves were normal and the lenses were not dislocated. The little patient was admitted to the hospital and observed for a number of days. Miotics succeeded in reducing the tension only to about 50, and on Nov. 1, 1917, trephining was performed on the right eye under ether.

The operation was made particularly difficult on account of the necessity of keeping the eye pulled down and the free bleeding which took place from the scleral tissues. After removal of the disk the prolapsed iris was excised. This was followed by an immediate presentation of vitreous. As it was feared that this presenting bead of vitreous would interfere with filtration, it was cut away, and there was a slight escape of vitreous which promptly ceased. The fold of conjunctiva was replaced and sutured and the flap lay perfectly flat. On coming out the ether the patient suffered from a good deal of vomiting. At the first dressing, Nov. 3d, a large swelling was found present ballooning up the conjunctival flap, evidently a prolapse of vitreous. The pupil was fully dilated and on inspecting the fundus, a white band of tissue with blood-vessels (the detached retina) could be seen drawn into the trephine-opening. Two days later the area of detached retina had enlarged and the prolapse of vitreous underneath the flap was also greater. There was no sign of inflammation. In the lower part of the fundus there was a red reflex, but light perception was lost. Gradually in the course of a few weeks the bulging underneath the conjunctival flap became smaller, the eye remained somewhat red, and recurring hemorrhages were observed in the vitreous. At present the eyeball is becoming phthisical, the conjunctival flap over the trephine-opening is flat, and the retina can still be seen drawn to the region of the trephine-opening.

The retrochoroidal hemorrhage in this case presumably occurred during the vomiting after the operation as the patient came out of ether, for the escape of vitreous from the trephine-opening had ceased when the conjunctival flap was replaced and sutured. The case is, furthermore, but another example of the dangers of general anesthesia in glaucoma operations, particularly trephining and sclerectomy. An additional complication was present in the subluxated lenses which caused the prolapse of vitreous into the trephine-opening. The patient's other eye presents the same condition, and

though the tension remains well above normal, the writer has not felt justified in advising an operation after the above-mentioned experience. If, however, an operation should become necessary, iridotaxis would seem to be the safest procedure.

# FOCAL INFECTIONS OF THE EYE FROM THE INTESTINAL TRACT.<sup>1</sup>

## A PRELIMINARY REPORT.

BY DR. J. G. DWYER, NEW YORK.

From Dept. of Laboratories, Manhattan Eye, Ear, and Throat Hospital,  
New York, N. Y.

**D**URING the last few years marked attention has been paid to the consideration of infections of various parts of the body, due to a low-grade chronic infection in other parts, especially the teeth and tonsils, and a very considerable advance has been made in the treatment of those so-called focal infections by the treatment of the original focus. The number of such cases on record is enormous, is fast increasing, and nowadays with certain infections we look for the primary cause and eradicate it if possible. Among these focal infections are some lesions of the eye, that used to be classified under a general classification as rheumatism etc. We know now that there is probably no such disease as rheumatism *per se*, but that the symptoms classified as rheumatism are probably manifestations of some focus of infection. The work on focal infections of the eye due to infections of the teeth and tonsils have been reported by many observers and need not be touched upon. This paper will deal with focal infections of the eye due in all probability to the absorption of toxins from the intestinal tract, and a few suggestions as to the method of treatment now being given a trial in order to overcome these infections, as, in comparison with the teeth and tonsils, it is

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<sup>1</sup> Read before the Section on Ophthalmology, New York Academy of Medicine, January, 1918.

impossible to treat surgically the intestine in the same way as the former two.

This investigation has been going on for about two years or more, and any statements that are made are based upon what we have learned in that time and are subject to change in the future, the whole idea of presenting the paper now being to direct attention to such infections and how possible to treat them.

At the beginning we were confronted with the fact that no two authorities were agreed as to what the intestinal contents should be in general—that is, the reaction, the amount of chemical constituents, the food detritus, and the bacteriology. This was and is a great difficulty and some may question our interpretation of what normal intestinal contents should be, but from an examination of hundreds of samples we have come to look for certain main characteristics, and I will begin by laying down what we look upon as the normal average intestinal contents.

First with regard to the reaction—Practically all authorities say that the normal feces is faintly alkaline to litmus. Now litmus is a rough and ready method of taking the titration, and we have found that with phenolphthalein the reaction is generally slightly acid.

Food detritus—Just as in diabetes and nephritis the modern schools of medicine are aiming to test the efficiency of the body to assimilate the various foods, fats, carbohydrates, and proteins, so here we examined the detritus, having these three fundamental bases in mind. Our guiding principle here was to cut out of the diet those types of food that were not being assimilated—in other words, indol, skatol, and phenol. These products are normally present and unless abnormal in amount can be disregarded, but as the former two are known to be absorbed into the system, appearing as indican in the urine, and as they are products of protein metabolism, we paid special attention to these, as will be noted later.

The bacteriology—Here we are confronted with the biggest problem of all, as the intestine normally contains many various forms of bacteria, but here we pick upon one outstanding fact. We made no endeavor to identify the various forms of bacteria present as that would be an impossibility, but bearing

in mind the work of various investigators, especially Metchnikoff, special attention was paid to the presence or absence of the colon bacillus. Now, the colon bacillus appears in the intestine a few days after birth and is usually found there throughout life. Again, the colon bacillus is Gram-negative—that is, staining a different color from that of the other bacteria usually found, these latter being Gram-positive with very few exceptions, and hence we had a ready means of comparison. The ptomaine producers are usually Gram-positive large bacilli, producing either acid or alkali, depending upon the group. Many of them are of the anaërobic varieties, such as the bacillus *aërogenes capsulatus* etc.

With these facts in mind and trying to narrow the whole affair down to a practical working basis, the following routine was carried out. The intestinal contents were examined as to their reaction, their content of indol and skatol, the food residue and the bacteria. Besides these cardinal principles, other side factors were noted. If the contents were found to be practically normal, these were noted as controls. Now in most of the patients examined, the contents could be classified under two headings, as to whether they were highly acid or alkaline. In both such classes the colon bacilli were either entirely absent or present only in very small numbers, the other bacteria being practically Gram-positive. This was true whether the reaction was to either extreme, as in a highly acid medium the colon bacillus cannot live, although it is an acid producer, and of course it is killed off in a highly alkaline medium. It was always found that in both highly acid and highly alkaline specimens the indol and skatol content was high and there was always a high percentage of indican in the urine. The following methods of treatment were then adopted: In the highly acid specimens, an endeavor was made to alkalinize the contents by the use of irrigation of 1% sodium carbonate solution, and then to practice colon transplantations in order to approach the normal. At the same time the patients were put on a rather free diet, cutting out however the food, such as meat, that was not completely assimilated—in other words, the tolerance for the three fundamental foods was established as far as possible. In the highly alkaline specimens, irrigations with sugar of milk were given and then the colon bacilli trans-

planted. At the same time, the Bulgarian bacilli were given by mouth, the whole idea being to approach the average normal. Sugar of milk was also administered by mouth in such cases to provide a suitable pabulum for the colon bacilli. At stated intervals, examinations were made and the treatment repeated until we got good growth of the colon bacillus.

A few of the eye cases will now be reported. The types of cases chosen were those that would not respond to any other form of treatment, cases that were thought to be focal infections, but treatment of the teeth and tonsils was unavailing. They were all cases that had lasted for weeks, better one day and worse another, but no real improvement.

CASE NO. 12.—Charles B., had lost the sight of the right eye on account of a low-grade ulcerative keratitis that had lasted six months and left the eye in a badly damaged condition with the pupillary area of the cornea covered with a scar. He had been treated for this eye by some of the best ophthalmologists in the city, and had been an in-patient of one of our hospitals for weeks. When I saw him, he had been in our wards for some weeks with the same condition in the other eye, the history of this lesion dating back two months. Every test had been negative and he was put on anti-specific treatment, as it seemed the most likely thing. Upon taking his history, we elicited the fact that he had had intestinal poisoning for years and could not get any relief, and he volunteered the information that the other eye had become quiet as soon as he had gone to a stomach specialist. The condition of the eye at first examination was as follows: A diffuse low-grade keratitis with a low-grade iridocyclitis; in spite of all measures, the pupil could not be kept dilated, but would dilate under the atropine and then contract again. One noticeable point was that the man's face was covered with an acne rosacea. Immediate examination of his intestinal contents was made and the following found: Reaction highly alkaline, indol and skatol five plus (+ + + + +) and bacterial smears totally Gram-positive. The treatment above outlined was carried out, and inside of two days the eye condition was much better, pupil remaining dilated under cycloplegic, and within a week he went home with a perfectly quiet eye. The acne cleared up and when he last reported, after six months, he was in splendid condition, with a perfectly serviceable eye.

CASE NO. 18.—J. Mc., a man suffering for nine months from a plastic iritis of low grade in both eyes, which kept



him from business and made a semi-invalid out of him. He had been under the care of competent ophthalmologists in the city, had had his teeth radiographed and the diseased teeth removed, had his tonsils out, and still the condition persisted. The same condition was found here, and within eight days the eyes had quieted down, and the last report I had, some months after the lesion, he was in excellent condition.

CASE NO. 19.—S. consulted me about his eye; he was suffering from an intense plastic iritis, that had lasted two months, and had had all the accepted methods of treatment. He cleared up rapidly with the treatment outlined for highly acid stools.

In conclusion we would close with a few references to the types of cases. By far the largest number of cases of choroiditis, retinitis, iridocyclitis, glaucoma, and such are diagnosed by us as to the condition present, but we cannot go any further in the etiology or in the treatment. The cases run their course in spite of the treatment adopted. It would be ideal, if we could take such cases and study them extensively so as to arrive at the cause. The reference of eye infections to the intestinal tract is not new, as evidenced by the splendid articles, among others of Herter and Smith published in 1894, reciting there cases of ophthalmoplegia, low-grade conjunctivitis, etc., cured by treatment of the intestinal tract. My attention was called to this article by Dr. Coffin, who incidentally was the means of my attention being directed to the intestinal tract on account of a case that he will describe in detail.

In conclusion, I would like to state that we don't claim anything new at all, it is simply an experiment along this line, but that the results in the series we have treated and which now number sixty-seven, were in the great majority of cases very striking, and it seemed well worth while to investigate along these lines. I wish to thank the men who have studied these cases with us and who placed them at our disposal, and to thank Mr. J. J. Connellan for the way in which he carried on the examinations.

THE LIGHT PUPILLARY REFLEX, ITS PATH, AND  
ITS ABOLITION CALLED IMMOBILITY OF THE  
PUPIL TO THE LIGHT REFLEX, AND REPORT  
OF A CASE OF UNILATERAL ARGYLL-ROBERT-  
SON PUPIL, IN WHICH CONSENSUAL REACTION  
EXISTED IN BOTH EYES.

By DR. ANTON LUTZ, HAVANA.

*(With eight figures, in the text.)*

A UNILATERAL Argyll-Robertson pupil must be considered even to-day as a rare anomaly. There exist, so far as I have been able to ascertain from the literature of the subject, only five cases of unilateral immobility to light with conservation of the consensual reaction in both eyes. The rarity itself would justify the publication of any new case of this last form. These cases are of the utmost importance in relation to the whole physiology and pathology of the pupillary reaction, and it will therefore be convenient to precede its publication by a short description of what we know to-day about the light reflex, its path, and its abolition called immobility of the pupil to the light reflex, or "Argyll-Robertson phenomenon."

I

DESCRIPTION OF THE LIGHT REFLEX.

As far as we know, the first exact modern description of the light reflex of the pupil, which was even known to Galen, dates from Verdier in 1751, who observed the contraction of the pupil on the incidence of light and who spoke of circular and radial muscles of the iris. The consensual light reflex or the

contraction of the pupil of the fellow eye dates from Whytt in 1752. Porterfield in 1759 called attention to the fact that the direct light reflex is stronger than the consensual one. Lambert in 1760 observed that the light reflex is stronger when the illuminated area is larger. E. H. Weber in 1852 observed that the macula has a stronger pupillo-motor effect than the periphery. Babinski, in 1900, noted that the adaptation of the retina has a pronounced influence on the amplitude of the light reflex. After keeping a patient for a certain time in a dark room, the light reflex can even be made visible in cases where it seemed to be abolished. The influence of the adaptation is so very strong that in normal persons a transitory anisocoria can be produced artificially by keeping one eye covered for a certain time.

The light reflex begins with the first day of life, even in prematurely born children. Magitot tells us that the light reflex begins at the end of the fifth fetal month. First, the pupillo-contraction reflex appears, in the direct as well as in the consensual; some weeks later the pupillo-dilatation reflex develops. In healthy persons it is not abolished through age. In old men, whose pupils are often miotic, it may be difficult to observe the contraction of the pupil; but we know to-day, through the systematic investigations of Winaver Bronislas on 180 old people from 60 to 95 years, that it is always present, and that its observation can be made easier by the use of a few drops of cocaine.

The light reflex consists in a contraction of the pupil when the illumination is increased, and further, in a dilatation of the pupil on a reduction of the illumination. In healthy persons incidence of light produces contraction of the pupil of the same side as well as of the pupil of the non-illuminated fellow eye. The contraction of the pupil does not show itself simultaneously with the incidence of light, but begins about 0.4 to 0.5 second later (Listing), and reaches its maximum after another one-tenth of a second. Vervoot (1900) showed its dependence on the quantity of light: an object as large as 1 illuminated with intensity 4 produces the same degree of pupillary contraction as an object 4 times larger but illuminated 4 times less. Ovio (1905) formulated the law in the following terms: the surface of the pupil is in inverse proportion to the square

root of the light intensity. Monochromatic light of different light intensity has also different pupillo-motor effect. Roentgen-rays have no influence on the pupil. The influence of light intensity is also shown in the consensual reaction: In healthy persons, upon covering one eye the diameter of the pupil of the fellow eye is increased from  $\frac{1}{4}$  to  $\frac{3}{4}$  mm. Schlesinger fixed as the threshold of the pupillary reaction a light intensity of 0.70 meter candle; this is normal for healthy persons of about 35 years of age; children have a little lower threshold; older adults a higher one. The pupils become tired, which is seen in a minor degree of contraction and the need of more light intensity and more time to produce the light reflex. The light reflex is always unconscious and involuntary. Hypnotic sleep (Janet) is without influence on the light reflex.

## II.

### THE PATH OF THE LIGHT REFLEX.

As far as we know to-day, the path of the light reflex may be divided as follows:

#### A. *Sensorial Motor Arc.*

1st. Sensorial afferent path, responsible for pupillary contraction:

Retina; optic nerve; chiasma; optic tract; corpus quadrigeminum anterius; fasciculus tecto-bulbaris.

2d. Efferent pupillo-contraction path:

Nucleus of the sphincter; nervus oculomotorius; radix brevis; ganglion ciliare; posterior ciliary nerves; the sphincter iridis muscle.

#### B. *Centers.*

Territory of the nucleus of the oculomotorius nerve.

#### C. *Sensory Motor Arc.*

1st. Sensory afferent path, responsible for pupillary dilatation:

All sensory cranial and spinal nerves with their correspondent spinal ganglions, especially the trigeminus, acusticus, and sensory nerves of the arms, chest, and neck.

2d. Efferent pupillary dilatation path:

Mesencephalon; medulla; lateral columns of the cord; ventral roots of the first three thoracic nerves; rami communi-

cantes; truncus sympathicus; superior cervical ganglion; anterior branch of the Gasserian strand; Gasserian ganglion; first branch of the trigeminus; long ciliary nerves; dilatator muscle.

The path of the light reflex is at present known only in a very uncertain manner, and it is very interesting to make a comparison of the different opinions and various clinical as well as anatomical statements concerning it. Let us follow it step by step.

#### *A. Sensorial Motor Arc.*

##### 1st. Sensorial afferent path.

It is obvious that the sensorial afferent path is responsible for pupillary contraction following an increase of illumination. It is doubtful if it can be made responsible for pupillary dilatation following a reduction of the same. It begins doubtless, in:

##### (a) *Retina.*

(aa) Most of the authors sustain that the whole retina is responsible for the light reflex, *e.g.* Lapersonne, Parsons,

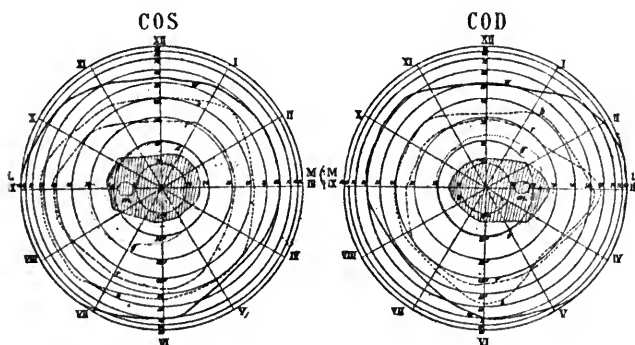


FIG. 1.—Campus pupillo-motoricus retinae (C. Hess).

Uthoff. Against this opinion C. Hess in 1907 brought forward the hypothesis, based on ingenious experiments that there exists a well marked pupillo-motor zone in the retina, which is alone responsible for the light reflex, and which includes the macula and a certain area around it about 3mm in diameter. Schlesinger believes this area to be 4mm in diameter. Heddaeus in 1886, stated that only the macula has a pupillo-motor effect.

Against the theory of Heddæus-Hess we have to take into account the cases with large central scotomata, as well as those with peripheric remnants of the visual field after inflammatory destruction of the optic nerve, in which the light reflex persists. Furthermore, against it we find that anatomically we do not know of any difference in the structure of the retina in this zone and of the remaining parts of the retina.

It remains certain that the macula has the strongest pupillo-motor effect, and that this effect decreases rapidly towards the periphery.

(bb) Most authors believe that the light reflex begins in the visual cells, *i.e.* in the rods and the cones. Uthoff expresses the opinion that the light reflex begins in the inner parts of the retina, and bases his idea on the fact that the light reflex is not always abolished through destruction of the outer retinal layers, *e.g.* in retinitis pigmentosa and detachment of the retina. But I think that in all such cases it remains uncertain whether the outer layers of the retina are in reality totally destroyed. The same observations induced Schirmer to see the origin of the light reflex in the so-called "amacrine cells of the retina," denying importance to the visual cells in respect to the light reflex. Against this opinion of Schirmer speaks the fact that the amacrine cells though abundant in the border of the macula are lacking in its center where we have the strongest pupillo-motor effect. The amacrine cells or "spongioblasts" have been specially studied by Ramón y Cajal. He gave them the name "amacrine cells" because they have no long axis-cylinder ( $\alpha$ =without,  $\mu\alpha\chi\rho\varsigma$ =large,  $\zeta$ =fiber); they are interposed on the one side between the centrifugal cylinder fibers which come down from the cortex and the basal ganglion to the retina, and on the other side between the large ganglion cells of the innermost retinal layer. Therefore we have to consider them as members of a centrifugal chain of neurons. In addition, between these amacrine cells are interposed other cells which have an ascendent axis cylinder ending in a knot in the external plexiform layer. We find that these last cells are in connection: first, by their inferior dendritic terminations with the centrifugal fibers as well as with the amacrine cells; and secondly, by their ascendent fibers with the articulations between cones and rods on one side,

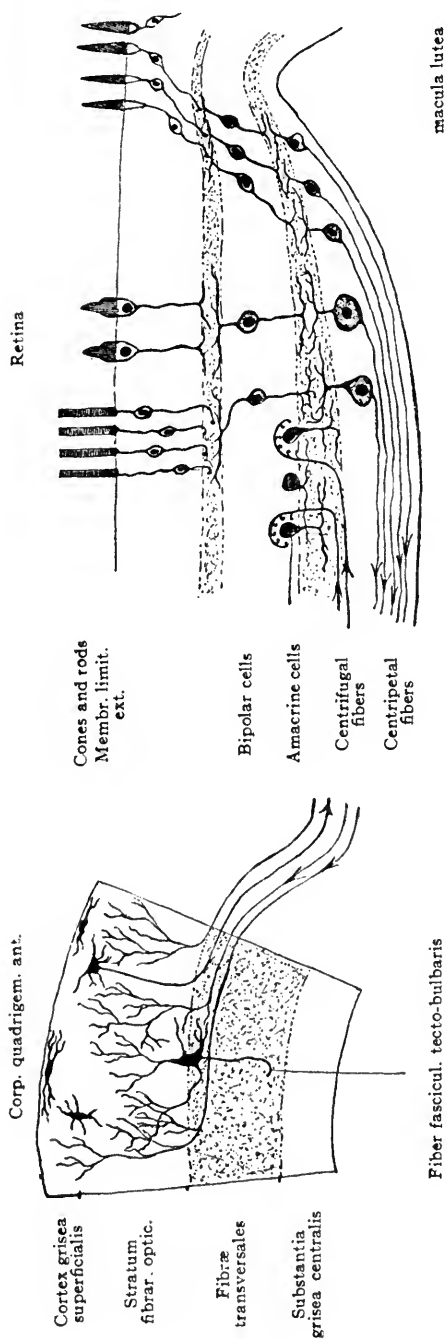


FIG. 2.—Sketch after S. Ramón y Cajal.

and the bipolar cells on the other side. According to Ramón y Cajal, these last cells belong to the centrifugal neuron chain, and complete the theory of Duval, which is the following: "The centrifugal fibers bring the stimulus down from the cortex and the basal ganglia, and produce a contraction of the ramifications of the bipolar cells and the ganglion cells; consequently, the surfaces of the dendrites come into a more intimate contact, the adaptation is more exact, and this facilitates the centripetal nerve current." This may be illustrated by a small sketch from Cajal.

Therefore, as far as our knowledge stands to-day, we have to accept that the amacrine cells belong to a centrifugal neuron system; if we consider further that these amacrine cells are found only in the territory of the second retinal neuron, corresponding to the spinal neuron of the spinal nerves, and that we never find them in the territory of the first retinal neuron, we cannot regard them as responsible for the origin of the light reflex. Lapersonne also believes that the amacrine cells play a rôle in the voluntary adaptation of the retina. We know that the amplitude of the light reaction depends upon the retinal adaptation, and we can admit that the amacrine cells play a rôle in the light reaction. The most plausible hypothesis on the difference of visual and pupillary fibers, is perhaps that set forth by Ramón y Cajal (1911): he called attention to the fact that in the innermost layer of the retina there are two different forms of ganglion cells; some are unistratified and are in connection with the shortest bipolar cells, which are connected with only one cone or one rod; this neuron chain is reserved for impressions of localization, and represents the individual path of the visual mental image to the brain. On the other hand, we have multistratified ganglion cells, which are connected with several multistratified bipolars receiving the impression of different cones and rods; these neuron chains would form the origin of the reflex path. In favor of this hypothesis is the embryological fact that the differentiation of rods and cones begins just at the end of the fifth fetal month, when according to Magitot's investigations on prematurely born children, the light reflex begins to develop. This theory would also explain the difference between the thick and the slender fibers in the optic nerve. It would also



serve to explain why the light reflex persists in cases of detachment of the retina, for example, after vision had been lost. Furthermore we have to remember that C. Hess in systematic experiments with light of different wave lengths, ascertained that not only the visual but also the pupillo-motor receiving apparatus is situated in the outer portions of the cones in day birds.

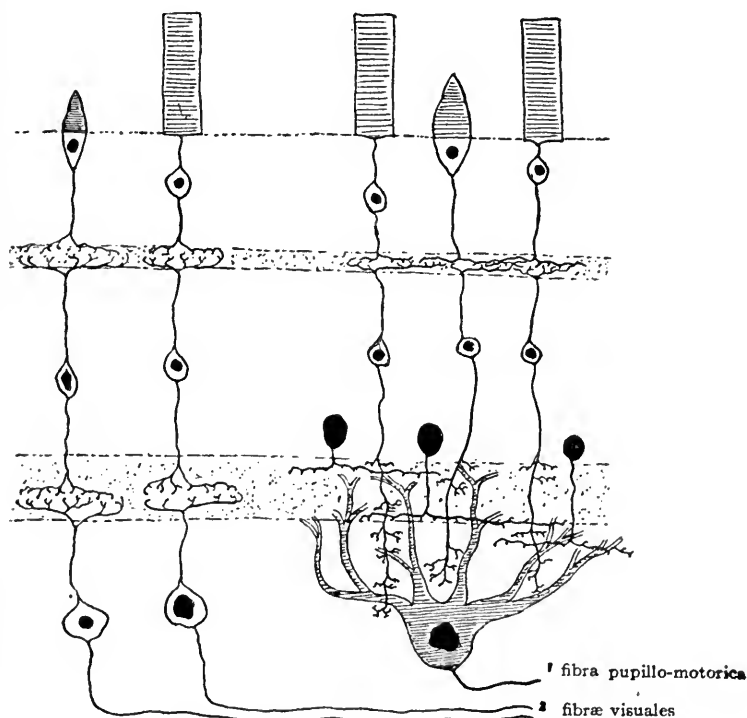


FIG. 3.—Sketch after S. Ramón y Cajal.

(b) *Optic Nerve.*

There is no doubt that there are both visual and pupillary fibers in the optic nerve. By cutting the optic nerve we obtain complete abolition of the light reflex (amaurotic light immobility). Key and Retzius could find in men two different kinds of fibers,—thick ones and slender ones. Westphal found that the thick fibers receive their medullary sheaths sooner than the slender ones. Monakow found that the thick fibers

passed almost entirely to the corpus quadrigeminum anterius, but not the slender fibers. It is therefore probable that the slender ones are concerned with visual sensation, and that the thick ones carry the afferent pupillo-motor impulse. But this is not yet proven, and authorities like Vennemann do not accept any special pupillary fibers. In favor of the acceptance of special pupillary fibers, we have to record the cases where we find light reaction of the pupil present, after complete loss of visual sensation, *e.g.* from inflammation of the optic nerve. Very interesting in this connection, is a case of Piltz, where the pupil contracted to light in a patient who had been completely blind for nine years from atrophy of the optic nerve. In compression of the optic nerve the pupillary reflex is much less affected than the vision, which may indicate that the pupillary fibers are more resistant. Furthermore the reaction of the pupil returns usually very much sooner than vision, when improvement begins after acute inflammation of the optic nerve. These considerations explain why we cannot accept the theory that the light reflex is only conducted by collaterals of the visual fibers—which does not seem improbable to Parsons; otherwise, visual sensation and light reflex should always be abolished simultaneously.

From purely theoretical considerations, in order to explain the light reflex, Ramón y Cajal investigated the chiasm for fibræ bifurcatæ. He readily found them there, and believes that they are responsible for the conduction of the light reflex. According to Henschen these fibræ bifurcatæ belong to the macular fibers. There are only a very small number of them, and they have not been found in man.

(c) *Chiasm.*

It is universally accepted that the pupillary fibers undergo a partial decussation in the chiasm exactly as do the visual fibers. This statement can be found in every book on ophthalmology or neurology. It is natural that this acceptance was suggested by the proven partial decussation of the visual fibers. Theoretically, we can consider three possibilities:

(aa) *Total crossing of the pupillary fibers in the chiasm:* This possibility is excluded by the clinical observations of heteronymous hemianopsia in which the pupillary reaction is almost never abolished. Mitchell published a case of complete

section of the chiasm by a tumor, where the pupillary reaction was present, nevertheless the chiasm was separated in two parts. It is further proven by the experiments of Bernheimer (monkey, 1898) and Bechterew (dog) where after having sectioned the chiasm in a sagittal direction, the presence of both the direct and consensual light reaction in both eyes could be demonstrated. These experiments and clinical observations, besides pointing towards partial decussation of the visual fibers, also tend to prove a partial decussation of the pupillary fibers, but do not exclude by any means the third possibility, *i.e.*, that the pupillary fibers do not undergo any decussation.

(bb) *Partial decussation of the pupillary fibers in the chiasm:* Parsons writes that this is proven by the hemiopic pupillary reaction of Wernicke. But it is well to bear in mind that Wernicke conceived the theory of the hemiopic pupillary reaction out of the hypothesis of a partial decussation of the pupillary fibers, and it is not logical to prove a hypothesis by its own unproven supposition. Harris records that in all lower animals (fishes,—lower mammalia) which are characterized by a total decussation in the chiasm, we have only a direct light reaction, but no consensual one, and that in all animals with partial decussation (higher mammalia—man), we have a consensual light reaction as well as a direct one. The rabbit has a partial decussation and a direct light reflex, but no consensual reaction. One could be tempted to conclude from these statements that the consensual reaction is founded on the presence of non-crossed pupillary fibers.

Most authors speak of the *hemiopic reaction of the pupil* (also called hemikinesis by C. Hess) with so much circumspection that one involuntarily gets the impression that these authors have never been convinced of the existence of hemikinesis. There are special methods necessary, as those of C. Hess and Clifford B. Walker, to avoid the disturbance through light dispersion and retinal adaptation, and to observe that most important condition of equikinetic parts of the retina. There are only a few physicians who have observed this reaction very clearly. Theoretically, we can find hemikinesis only in hemianopsia anterior, *i.e.* through total destruction of the optic tract between chiasm and corpus quadrigeminum

anterior. This space is perhaps only one fifth of the length of the whole optic tract from chiasm to cortex. This proportion, and the fact that most hemianopsiæ are produced by hemorrhages in the internal capsule or in traumatism of the occiput, explain the small number of published cases of hemianopsia anterior. Traumatism in this region in most cases kills the patient. Lenz (1909) gathered all the cases of hemianopsia, and tells us there did not exist a definite case of complete section of the optic tract in which microscopical examinations had been made. A. Jess (1913) published from the clinic of Würzburg an article on the practical value of hemikinesis, which he considers, on account of one positive case, as a sure diagnostic aid in the localization of brain affections. The positive case of hemikinesis which he reports was followed by an autopsy. In the report he only mentions right hemianopsia with preservation of the macula, choked disk in both eyes, and right hemiparesis involving the facial, hypoglossus, and trigeminal nerves; there is nothing said as to whether the section of the optic tract was complete and the preservation of the macula speaks in favor of an incomplete section. Lenz (1914) published later another article on hemianopsia and reported six cases of hemianopsia anterior: one case could not be examined for hemikinesis on account of immobility of the pupil to the light reflex in both eyes, from syphilis; in two cases he found no hemikinesis, the reaction was normal; in three cases there was hemikinesis, and of these three only one showed a complete hemianopsia, though there was a little remnant in the abolished visual field near the macula. The two others were cases of hemianopsia for colors: only (hemiachromatopsia), which Lenz considers as lesions of minor severity. It is, therefore, all the more surprising that Lenz could find hemikinesis, for we must admit that real hemikinesis should only be possible in complete destruction of the optic tract which conducts the pupillary fibers. These observations of Lenz are in conformity with the systematic researches of Clifford B. Walker, who examined fourteen cases of hemianopsia in the clinic of Cushing (1914). He made the tests with all precautions and with a specially built apparatus; he could find hemikinesis present in anterior hemianopsia as well as in posterior. It was found in cases having

every clinical evidence of being purely posterior cases; one of the most striking examples was a case of posterior hemianopsia. He also found it in rather early stages of incomplete hemianopsia of both bitemporal and homonymous types. The most important fact is that hemikinesis was present in a completely normal eye, after having lowered the adaptation of one half of the retina (one half of the retina was blinded by looking at the edge of a large bright surface and a difference in the pupillary reaction with the rotatory shutter was obtained). One can conclude from these observations of Walker that the adaptation of the retina is of the utmost importance for the light reflex, but we must also conclude that the hemiopic pupillary reaction is such an uncertain and scarcely known phenomenon that it can be by no means a proof for such an important thing as semi-decussation of the pupillary fibers.

The observations of Clifford B. Walker are confirmed by Déjerine and Jumentié (1914) who observed a case of hemianopsia posterior followed by autopsy, in which both observers had clearly noted the difference in the pupillary reaction of the healthy, and the injured, visual side. Another point we have to consider is the following: In publications on hemikinesis we find mostly positive hemikinesis without any note of whether it was found in both eyes, and to the same degree. Theoretically we have to assume that it would be so in every case; further clinical observations are necessary for the evidence to be considered conclusive.

In favor of a partial decussation of the pupillary fibers in men, we have the experiments of Trendelenburg in cats (1911); by cutting one optic tract in a cat he could observe hemikinesis and mydriasis of the contra-lateral eye. But regarding the fact e.g. that lower animals have total decussation, it seems to me not allowable to conclude out of these experiments that we have semi-decussation of the pupillary fibers in the chiasm in man. Much more convincing is a case of anterior hemianopsia observed by Pierre Marié and Ch. Chatelin (1915); lesion of the right tract by a piece of metal, left hemianopsia observed for one year, limiting line going exactly through the point of fixation and left pupil more dilated. Examination for hemikinesis made with all precautions at different times showed: "Right eye: if the light falls on the blind half of the

retina, the contraction of the pupil is good but does not persist; if it falls on the seeing half of the retina, the reaction is normal. Left eye: if the light falls on the blind half, no pupillary contraction follows: if it falls on the seeing half, the contraction is good but does not persist." These observations show that the hemikinesis is not equal in both eyes, and one could be tempted to deduce that most pupillary fibers undergo semi-decussation, consequently hemikinesis is more pronounced on the contralateral side, because the contralateral eye has lost the nasal retina, which includes a larger part of the pupillomotor zone (area between disk and fovea) than the temporal retina. But such deduction would be in contradiction of the fact that in hemianopsia bilateralis, the pupillary reaction shows no alteration and that sagittal cutting of the chiasm remains without influence on the light reflex. In addition it is good to bear in mind that there exists a certain variation in men, in the degree of the chiasm decussation of the visual fibers. There are observations of total crossing of the chiasm as well as of congenital absence of any chiasm crossing (Cushing and Walker, *Brain*, 1914).

Ramón y Cajal showed by a very ingenious theory that lower animals have a complete crossing of the visual fibers, and that higher animals have only a partial decussation,—to make possible simultaneous vision. If this theory is true, there is no necessity for semi-decussation of the pupillary fibers.

We have therefore to consider a third possibility:

(cc) No crossing of the pupillary fibers in the chiasm: If we ask ourselves, first if there exists any fact which would render impossible the admission of this non-decussation, then we must admit that so far there is no convincing fact reported which would permit us to discard absolutely this view. What would be the consequences of such a statement? There are two: first, in cases of binasal hemianopsia, we should have a complete abolition of the light reflex; and, secondly, in cases of complete destruction of the tract, we should get homonymous hemianopsia with pure light immobility of the pupil of the same side. Lange published (1913) as far as I know the first well-studied and surely recognized case of hemianopsia binasalis: it was a young man in whom neurological examina-

tion could not bring out an indication of any abnormality and who showed a complete hemianopsia binasalis for three fourths of a year, together with an external squint which is often present in reduced vision, and who further had—what is most important—a complete abolition of the pupillary reflexes for light and convergence. Now it seems to me quite natural that the reaction on convergence is abolished, since there is no object for it; it is well known how difficult it is for blind people to converge when we do not touch their nose, and so on. The most striking fact was that the pupils did not react to light, the nasal retina in both eyes being quite intact, and vision reduced to fingers at 4m. A technical error can be excluded in the investigations of such an experienced oculist as Lange. We have two further observations on binasal hemianopsia with indications on the light reflex, but these two cases are not without complications. Charles R. Heed and George E. Price (1914) observed one case which they considered as a tabetic manifestation; the binasal hemianopsia was present for three months, the direct as well as the consensual light reflex being abolished in both eyes, the reaction on accommodation being sluggish; there existed slight Romberg, positive Biernacki, loss of Achilles tendon reflex, and lymphocytosis of the cerebrospinal fluid. Shoemaker (1905) described a case of binasal hemianopsia; the pupils were equal and the irides reacted rather sluggishly to light, Wernicke being imperfectly present. The second consequence would be, as mentioned before, a pure light immobility of the same side. As no certain case of complete destruction of the tract is on record we do not know anything about it, pro or contra. Hemianopsia visualis and pure light immobility of one eye would then be the symptoms of the complete destruction of the tract. As far as I know, these have not been observed, and further clinical observations are required. With these considerations I do not wish to affirm that there does not exist decussation of the pupillo-motor fibers in the chiasm. I wish only to show that we cannot exclude the possibility of non-decussation, as long as the semi-decussation is not absolutely proven, and that in further anatomical researches and clinical observations, we have to take in consideration the possibility of non-decussation in the chiasm. We can therefore never say that the partial

decussation of the pupillo-motor fibers in the chiasm is proven by the hemikinesis; we can only suppose that it is probable by analogy with the visual fibers. Only after having given proof that non-decussation of the pupillary fibers in the chiasm is excluded, can we accept by exclusion the semi-decussation of the pupillary fibers in the chiasm.

(d) *Optic Tract.*

Opinions are also divided as regards the optic tract (Figure 4).

This sketch shows the most accepted description of the path of the light fibers in the tract. According to Ramón y Cajal, v. Monakow, Van Gehuchten, Testut, etc., the pupillary fibers follow the optic tract as far as the corpus geniculatum externum. There, the visual fibers separate from the thick pupillary fibers, the visual fibers going into the corpus geniculatum externum, and pulvinar, and only very few pass through the corpus geniculatum to the tuberculum quadrigeminum anterius. The pupillary fibers leave the tract in front of the corpus geniculatum externum and go in their totality to the corpus quadrigeminum anterius through the brachium conjunctivum. This is proven to-day (1913) by the experiments of Karplus and Kreidl who cut in a monkey and in a cat both brachia conjunctiva and corpus quadrigeminum anterius; the animals survived the operation about one month and during that time the light reflex could not be detected, while the reactions on convergence and on pain were normal. Bechterew believed (1883) that the pupillary fibers leave the tract just behind the chiasm, entering the stratum griseum, and that they reach, following the bottom of the third ventricle, the oculomotor nucleus. He was led to this opinion by the existence of the ganglion opticum basale (Meynert-Huguenin), which is a conglomerate of ganglion cells in the chiasm. On each side of the chiasm is one ganglion which is in connection with the retina of the same side, and whose continuation is found in the tuber cinereum. This neuron would therefore represent a direct or non-decussated reflex path in the optic nerve. Trendelenberg (1911) could cut these latter fibers in cats, but the pupillary reaction did not show any alterations afterwards. Bechterew gave up this idea later, sharing those of Van Gehuchten (1900) that the fibers leave the tract before the



corpus geniculatum externum, and enter then into the posterior part of the thalamus and reach the oculomotor nucleus

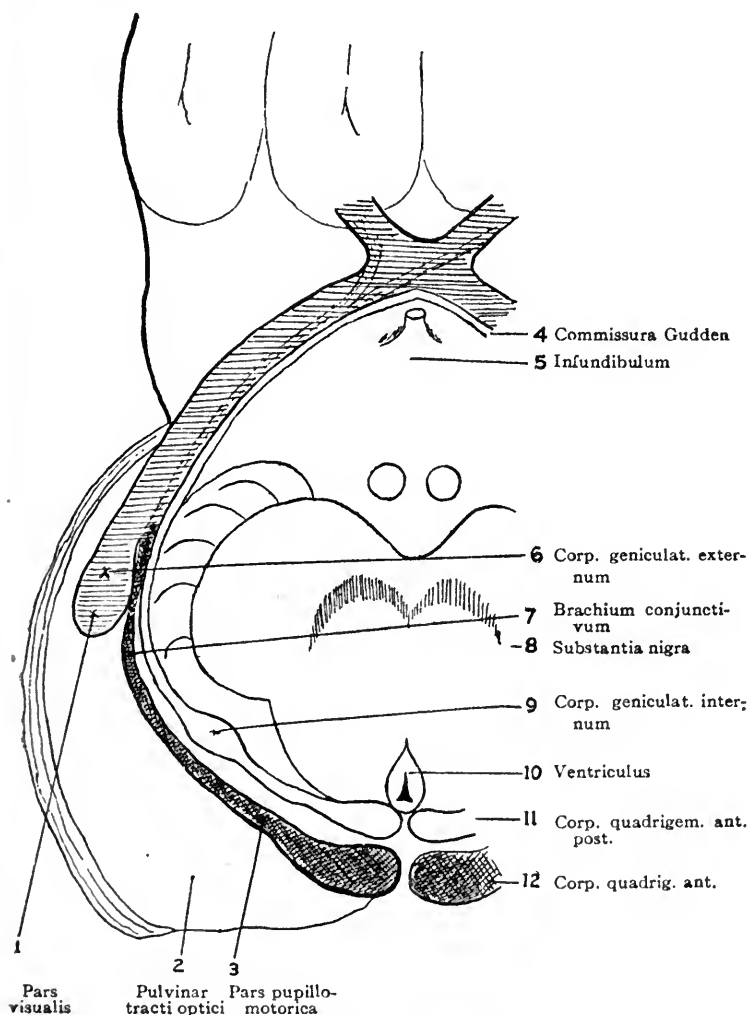


FIG. 4.—Sketch after Testut.

through the commissura blanca posterior. After the description of Déjerine (1914) these fibers originate in the pulvinar. Bernheimer (1899) concluded from embryological studies that the pupillary fibers go in part direct from the tract through the

corpus quadrigeminum to the nucleus, while other parts enter in connection with the gray matter of the corpus quadrigeminum. Against the acceptance of this view stand the systematic investigations of Probst, who never could find that the degenerations following enucleation of eyeballs go further than the corpus quadrigeminum antierius. Therefore, we have to accept that in the corpus quadrigeminum antierius begins a new neuron.

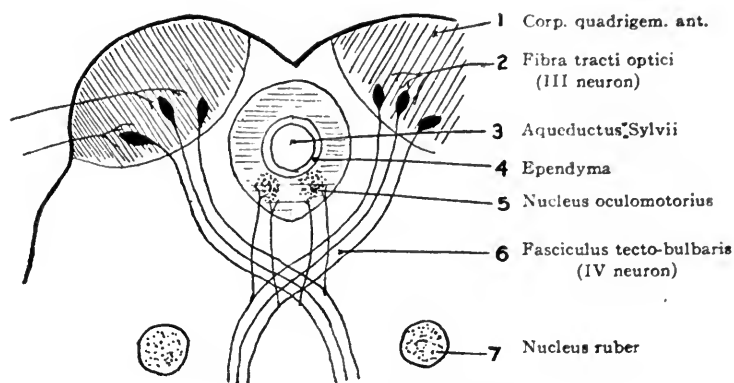


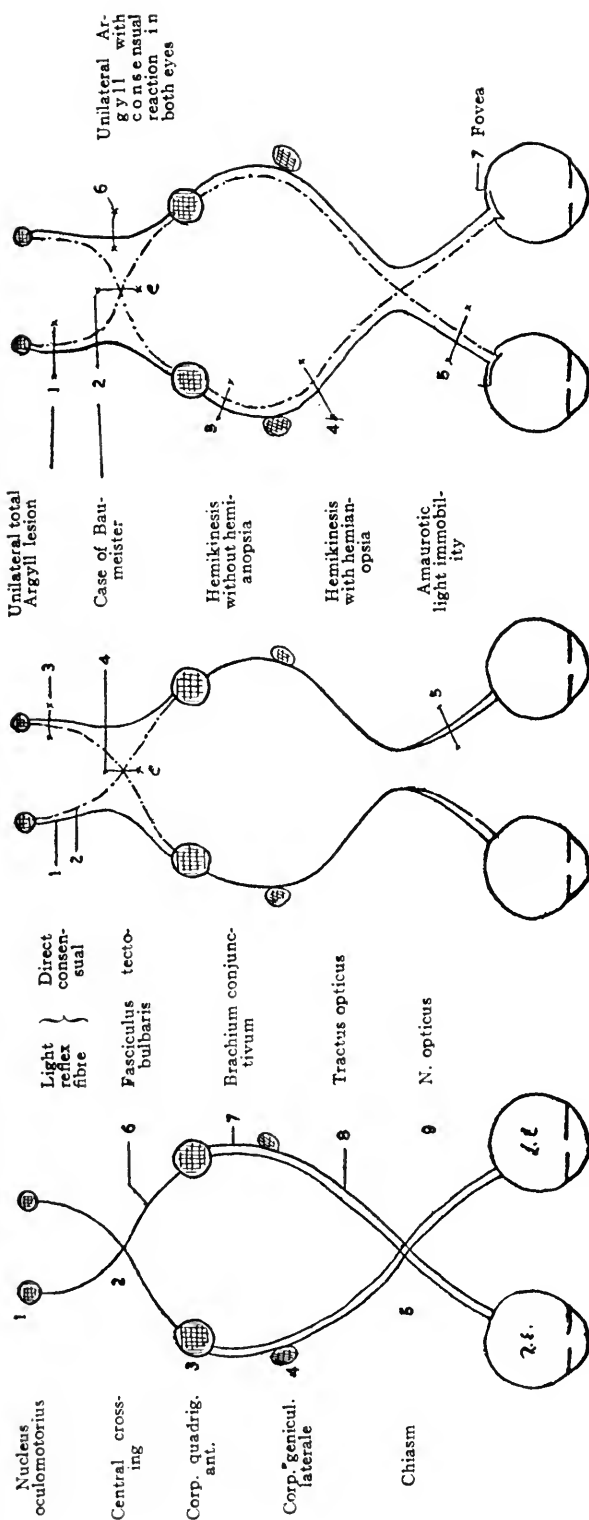
FIG. 5.—Sketch after Testut.

(e) *Corpus quadrigeminum antierius.*

Van Gehuchten (1908) believes that the corpus quadrigeminum antierius is the reflex center, because it does not give origin to ascendent fibers. Others, like Testut, accept ascendent fibers, but nevertheless consider the corpus quadrigeminum antierius as the reflex center. Van Gehuchten as well as Testut believes that the pupillary fibers go to the ganglion cells of the corpus quadrigeminum antierius and enter there in connection with the fasciculus tecto-bulbaris.

This fasciculus descends from the top of the mesencephalon, goes around the aqueduct of Sylvius, across then to the other side, and enters in the fasciculus longitudinalis posterior; in its path the fasciculus tecto-bulbaris gives collaterals to the ganglion cells of the nucleus oculomotorius of both sides, as well as to the other motor nuclei which play a rôle in the movements of the eyeball.

Ramón y Cajal (1911) gives the following description of this



III. Semi-decussation of the pupillary fibers in the chiasm.

FIG. 6.

last part of the afferent pupillo-motor path. He says: "The third retinal neuron passes with its axis cylinder to the tuberculum quadrigeminum anterius: there it enters into connection with the fibers which form the commissura blanca cerebri posterior, which is situated just in front of the corpora quadrigemina anteriora. These fibers then pass down either directly or after crossing to the other side, and enter into connection with the nucleus interstitialis, which is the origin of the fasciculus longitudinalis posterior; this fasciculus longitudinalis posterior is the last stage, because it brings the light stimulus to the nucleus of the oculomotor nerve as well as to the other nuclei which are responsible for the motility of the eye."

The communication between the corpus quadrigeminum anterius and the oculomotor nucleus takes place through the fasciculus tecto-bulbaris, and according to Testut through the commissura blanca posterior and the nucleus interstitialis according to Cajal. We have to consider the possibility that the first could be the path for the direct light reaction, and the second the path for the consensual light reaction. The first one has only one neuron interposed, the second has two. For this reason it seems to me more correct to make the fasciculus tecto-bulbaris responsible for the light reflex, and the route through the commissura blanca posterior, for the reflex movements of the eyeball, *e.g.* when a visual impression falls on the periphery of the retina. This opinion is confirmed by the experiments of Harriss (1903), who made two sections through the commissura blanca posterior, with the following result: In the first three days a marked mydriasis was noticeable; this disappeared on the fourth day, after which there was a completely normal reaction.

It seems natural to me to make responsible for the consensual light reaction, collaterals of the pupillary (not visual) fibers for the direct light reaction.

These considerations show us how little known is this last part of the afferent pupillo-motor path, but there is another point to which we have to call attention: it is the partial or total decussation of the pupillary fibers in this last stage of the afferent path. The efferent pupillo-motor path does not undergo any decussation and the center is situated in the oculomotor nucleus. Lower animals show a total crossing of

the optic nerves; they have a direct light reaction, but no consensual one. So we are forced to accept as a consequence of the total crossing in the chiasm a second total crossing before the afferent path reaches the center; if that were not so, that is to say, if we had no central crossing, we could only have a consensual reaction, but never a direct light reaction. Applying the same considerations to animals with partial decussation of the visual fibers in the chiasm there may be different arrangements of the pupillary fibers in the chiasm.

A satisfactory explanation of the pupillary reaction can only be given in accordance with the last sketch, in which we find a partial decussation of the chiasm and semi-decussation in the center, showing that each half of the retina is in connection with both sides of the photomotor nucleus. A mere glance at the preceding sketch shows how simply the connections of the afferent pupillo-motor path with the center takes place, if we do not accept semi-decussation of the pupillary fibers in the chiasm.

A comparison of these different opinions on the route of the light fibers appears to me to be very important. From them we draw the logical conclusion, that, by lesions in different parts, the following possibilities may occur:

(A) A lesion between corpus geniculatum laterale and corpus quadrigeminum anterius causes:

1st. Hemikinesis without hemianopsia, if we accept semi-decussation of the pupillary fibers in the chiasm. This phenomenon has, as far as I know, not yet been observed clinically.

2d. Abolition of the direct light reflex in the eye of the same side, and abolition of the consensual reaction in the eye of the other side, if we accept a non-decussation of the pupillary fibers (amaurotic light immobility).

(B) A lesion between corpus quadrigeminum anterius and the photomotor nucleus leads to abolition of the direct as well as consensual light reflex in the eye of the same side, whether we accept a non-decussation or partial decussation of the pupillary fibers in the chiasm. This phenomenon has been observed several times clinically, and represents in cases of lesion on one side, the unilateral Argyll-Robertson phenomenon. As the Argyll-Robertson phenomenon or immobility

to the light reflex is generally found bilaterally, we have to look for the crossing of the pupillary fibers in the mesencephalon, not in the neighborhood of the corpora quadrigemina but in the neighborhood of the photomotor center below the aqueductus Silvii, where a lesion of small dimension can interrupt the communication of the afferent pupillo-motor path on both sides with the photomotor centers of both sides. A lesion in the point marked C in the sketch of the central crossings must lead to a suppression of the consensual reaction in both eyes. We would then find patients who had direct light reaction in both eyes but no consensual reaction. As far as I know, there exists only one observation of this kind in literature, namely, that of Baumeister (*Archiv f. Ophthalmologie*, xix.); he saw a man who showed direct light reaction in both eyes whilst the consensual reaction could not be detected in either.

Based on the above description we have as chain of the neurons of the afferent pupillo-motor path:

- |                                     |          |
|-------------------------------------|----------|
| 1. Cones and rods.                  | } RETINA |
| 2. Multi-stratified bipolar cells.  |          |
| 3. Multi-stratified ganglion cells. |          |

whose fibers are found as thick ones in:

Optic nerve,  
Chiasm,  
Optic tract,  
Brachium conjunctivum.

- |   |                  |
|---|------------------|
| 4. Ganglion cells of the deeper layers of the corpus quadrigeminum anterius whose fibers form the fasciculus tectobulbaris. | } MESENCEPHALON. |
| 5. Ganglion cells of the photomotor center in the oculomotor nucleus.   |                  |

2d. *Center for pupillo-contraction.*

As the centers are interposed between the arcus sensorio-motoricus as well as between the arcus sensitivo-motoricus, it may be convenient to describe them in part together with the arcus sensorio-motoricus, and in part together with the arcus sensitivo-motoricus.

If we have to consider as the center the point where a centripetal impression changes into a centrifugal reaction, then it is easy to understand that we have to consider as center for pupillo-contraction, or photomotor center, the oculomotor nucleus, by which the excitation received by the retina returns to the periphery (iris).

There are three different opinions about the situation of the photomotoric center:

(a) Majano and Van Gehuchten place it, as Flourens did in 1824, in the nucleus lateralis of the corpus quadrigeminum anterius, because it does not give origin to ascendent fibers; this hypothesis has been attacked, especially by Bechterew. We must note here further, the experiments of Gudden, who removed superficial parts of the corpus quadrigeminum anterius, without subsequent changes in the pupillary reaction.

(b) Bernheimer places it in the mesial nucleus with small ganglion cells described by Edinger and Westphal, and bases his opinion on the following experiment: In a monkey he excited this nucleus electrically, producing pupillo-contraction; in another monkey, he destroyed this group of ganglion cells and abolished the light reflex; further he found degenerations in this part of the oculomotor nerve after enucleation of the eyeball. These last statements could not be verified by Probst.

Against the first two arguments we have to say that it is almost impossible to excite or to destroy the nucleus of Edinger-Westphal alone, without injuring the neighboring portion. It is too near to the nucleus principalis lateralis. Ramón y Cajal emphasizes the fact that he, as well as many other investigators, could never see that fibers from the ganglion cells of the nucleus Edinger-Westphal join the roots of the oculomotor nerve; he clearly gives expression to his opinion that the nucleus Edinger-Westphal has nothing to do with the oculomotor nerve.

(c) v. Monakow believes that the whole gray matter around the third ventricle is responsible, as well as ganglion cells scattered in different parts of the nucleus oculomotorii, and especially the frontal and dorsal pole of the ventral part of the chief lateral nucleus. He is supported in this by Bach and Tsuchida. They base their opinion on the fact that they found intact the nucleus of Edinger-Westphal in cases where

the light reflex was abolished, and that in other cases where this part was completely destroyed, the light reflex was present. The hypothesis of Monakow is strongly supported by the embryological fact that in the fifth fetal month in which the light reflex begins, the chief lateral nucleus alone is developed, and that the nucleus of Edinger-Westphal develops at the earliest in the seventh month. Against the acceptance of the nucleus Edinger-Westphal as the photomotor center, we have further to mention that it is only found in men and monkeys, but not in other animals.

Another important point to which we have to call attention is that the fibers which leave the photomotor center and which must be made responsible for the light reflex—that is, the first step of the efferent pupillo-contraction path, do not undergo any decussation in the pedunculus cerebri. In this regard they differ fundamentally from the fibers which go to the external muscles of the eyes. It is important to decide this point; if further investigations should not show a decussation in this first stage of the efferent path, we are logically forced to accept a semi-decussation of the afferent pupillo-motor fibers in the space between corpus quadrigeminum anterius and nucleus photomotoricus. If this decussation of the afferent pupillo-motor path should not exist, we would not be able to explain the existence of the consensual light reaction. For, as I showed before, the semi-decussation of the chiasm alone does not explain its existence.

3d. *Efferent pupillo-contraction path.*

It was Herbert Majo who called attention (1823) to the fact that pupillo-contraction depends on the integrity of the oculomotorius. By dividing this nerve we can abolish the light reflex; also degeneration of its nucleus produces the same effect. It was Claude Bernard who showed that, notwithstanding the abolition through cutting of the nerve, it was not possible to produce pupillo-contraction by electrical excitation of the nerve in its intracranial path (1860); to produce contraction it was necessary to excite the ganglion ciliare or the nervi ciliares breves. This fact demonstrated to him the importance of this ganglion. Only the fibers for pupillo-contraction and for accommodation enter the ganglion ciliare and find there the connection with the small ganglion cells.



Langley and Anderson showed that after application of nicotine to the ganglion ciliare it was no longer possible to produce pupillo-contraction through electrical excitation of the radix brevis ganglii (1892). Apolant showed (1896) that after having

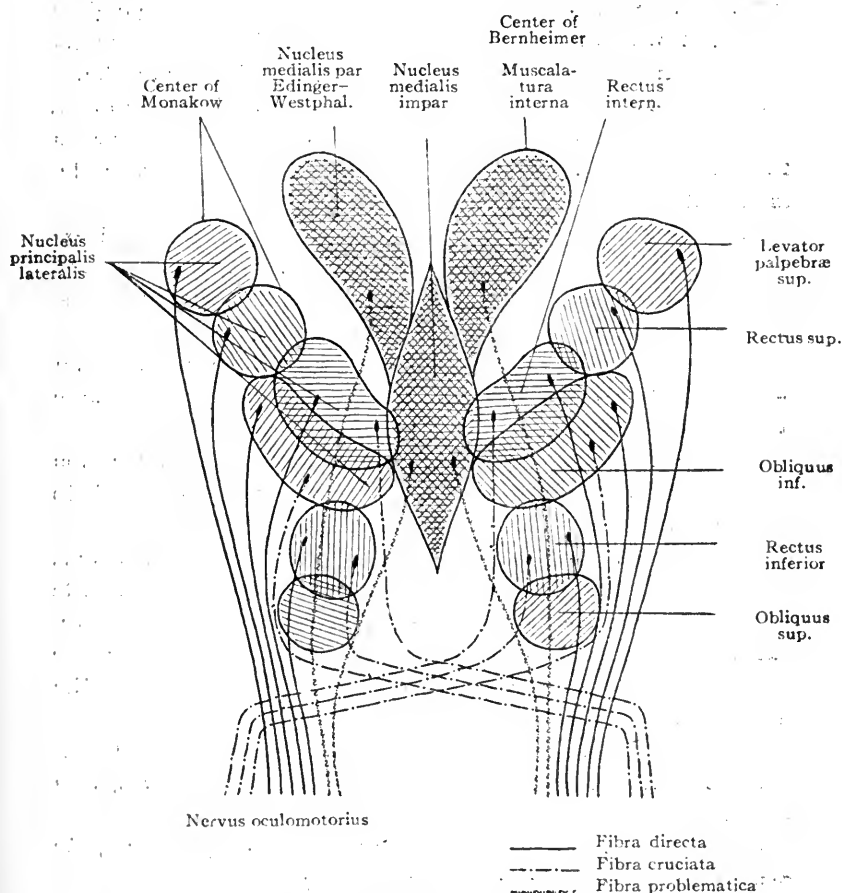


FIG. 7.—Sketch after Testut.

cut the nerve within the skull, the degeneration of the fibers extends only as far as the ganglion, and does not invade the nervi ciliares breves. Angelucci and Lodato showed (1905) that the degeneration, after destruction of the oculomotorius, remains restricted to the small ganglion cells. Langendorff showed that, after having killed a cat, excitation of the nervus

oculomotorius remained without effect on the exterior muscles of the eye, but that it was possible to produce irido-contraction within a certain time after death, by electrical stimulation of the nervi ciliares breves. Frank showed that after having cut the radix brevis, longa, and sympathica of the ganglion ciliare, it was yet possible to produce irido-contraction by stimulation of the sensory nervi ciliares, and that by mechanical compression of the ganglion it was possible to suppress the said pupillary reaction. This shows the importance of the ganglion ciliare and its independence. It is further demonstrated by the following observations:

(a) Brown Sequard enucleated the eyes of frogs and exposed them alternately to light and darkness, and thus obtained irido-dilatation and irido-contraction, from 50 to 100 times.

(b) Clinical observations show us, in very rare cases, that we can produce irido-contraction through focal illumination in absolutely blind eyes.

(c) Hertel (1906) could produce this irido-contraction through systematic experiments on blind eyes, with the help of an electrical arc light (ultra violet rays).

It is uncertain whether these three above-named irritations pass through the ganglion ciliare, or act locally on the muscle cells of the sphincter iridis. It also shows with what precautions we should accept such cases as arguments for the existence of separate pupil and visual fibers. Cutting of the nervi ciliares breves abolishes pupillary contraction.

Excitation of the short ciliary nerves produces rapid contraction of the pupil; excitation of the long ciliary nerves, slow dilatation.

Considering these facts we have the following: *chain of the neurons in the efferent pupillo-contraction path*:

1. Ganglion cells in the nucleus photomotoricus, whose fibers form part of the nervus oculomotorius.
2. Small ganglion cells in the ganglion ciliare, whose fibers form nervuli ciliares breves.
3. Muscle cells of the musculus sphincter iridis.

B. *Sensory motor arc.*

1. Sensory afferent path.

It is obvious that this part is responsible for pupillo-dilata-

tion. The excitations come from all sensory nerves of the entire body—from the spinal nerves as well as from the cranial. But of special importance are:

1st. The spinal nerves of the last cervical and of the first three thoracic segments; that is, skin of the neck, of the shoulders, of the arms, and of the breast.

2d. Nervus trigeminus; skin of the face.

The section of the trigeminus produces an insensibility of the iris and a slight contraction of the pupil, which develops slowly and lasts half an hour (suppression of the inhibitive influence of the trigeminus through fibra ascendens fasciculi longitud. post.). This contraction becomes permanent through section of the nervus ophthalmicus (cutting of the sympathetic fibers for the long ciliary nerve). Electrical excitation of the peripheral part of the ophthalmic (Caillaud, 1907) produces a yet stronger contraction of the pupil. Irritation of the sympathetic long ciliary fibers. This shows that we have another sensory reflex arc through the ganglion Gasserii. Enucleation of the eyeball in monkeys is followed by degeneration of a certain number of ganglion cells in the ganglion Gasserii (Marina).

2. Center for pupillo-dilatation.

It is not certain that there really exists a center for pupillo-dilatation in the mesencephalon. We could expect it from the fact that lowering the illumination produces dilatation of the pupil. It is probable that there does not exist an anatomical well-circumscribed part of the brain for such a center, but that this dilatation center is a physiological or hypothetical one and that we understand by it, all the inhibitions which come from the periphery and from the cortex and which influence the photomotor center for pupillo-contraction. The inhibitive influence of the cortex is shown by the fact that we find a small pupil in all cases where these cortical inhibitions are eliminated: in the newly born child, where the cortex is not yet developed, in old people where the cortex is atrophied, in sleep, and in slight narcosis where the excitations coming from the periphery are not elaborated in the cortex, and also as a consequence of morphine, by its narcotic action on the cortex, and finally, in syncope. On the other hand, we observe the influence of the cortex in the very large pupil, upon awaken-

ing, or in darkness where the cortical stimuli get the upper hand over the light excitations.

Bechterew called attention to the fact that pain sensation not only produces pupillo-dilatation, through excitation of the centrum cilio-spinale, but also by inhibition of the center for pupillo-contraction. This inhibition of the photomotor pupillo-contraction center was demonstrated by Braunstein, who showed that excitation of the sciatic nerve was followed by pupillo-dilatation even after complete section of the cervical sympathetic. Even after cutting the trigeminus, the ganglion cervicale superius, and the cervical cord, frightening the animal produced irido-dilatation. This experiment can be explained only through a pure cortical inhibition. But there also exists a pure reflex dilatation without any influence of the cortex, as shown by the experiments of Trendelenburg, who produced pupillo-dilatation after extirpation of both hemispheres.

We know very little of the connection of the afferent sensorio-pupillo-motor pathway, with its efferent pupillo-contraction path. We know almost nothing about the connection of the sensorial as well as the sensory afferent pupillo-motor pathway with its efferent pupillo-dilatation path. Pupillo-contraction is executed through the para-sympathetic vegetative nerve of the mesencephalon—*i.e.*, the nervus oculomotorius; pupillo-dilatation is produced by the sympathicus, and it is natural to conclude that the origin of the pupillo-dilatation path is the sympathetic vegetative nerve of the mesencephalon which runs down from the corpora quadrigemina through bulbus and medulla to the cervical cord. It is a very surprising fact, that excitation of the opticus produces pupillo-contraction, while excitation of all other sensory nerves, cranial as well as spinal, is followed by pupillo-dilatation. For the connection of these different sensory nerves with the photomotor contraction center, or its afferent sensory path, we have to consider the fasciculus longitudinalis posterior. This fasciculus longitudinalis posterior goes down from the mesencephalon to the spinal cord. The fasciculus longitudinalis is situated so near to that of the other side that they come in contact in some places. The fasciculus is composed of:

(a) *Fibræ ascendentes.*

These are direct fibers which conduct the excitation from the ganglion cells of the posterior horns, through the tractus

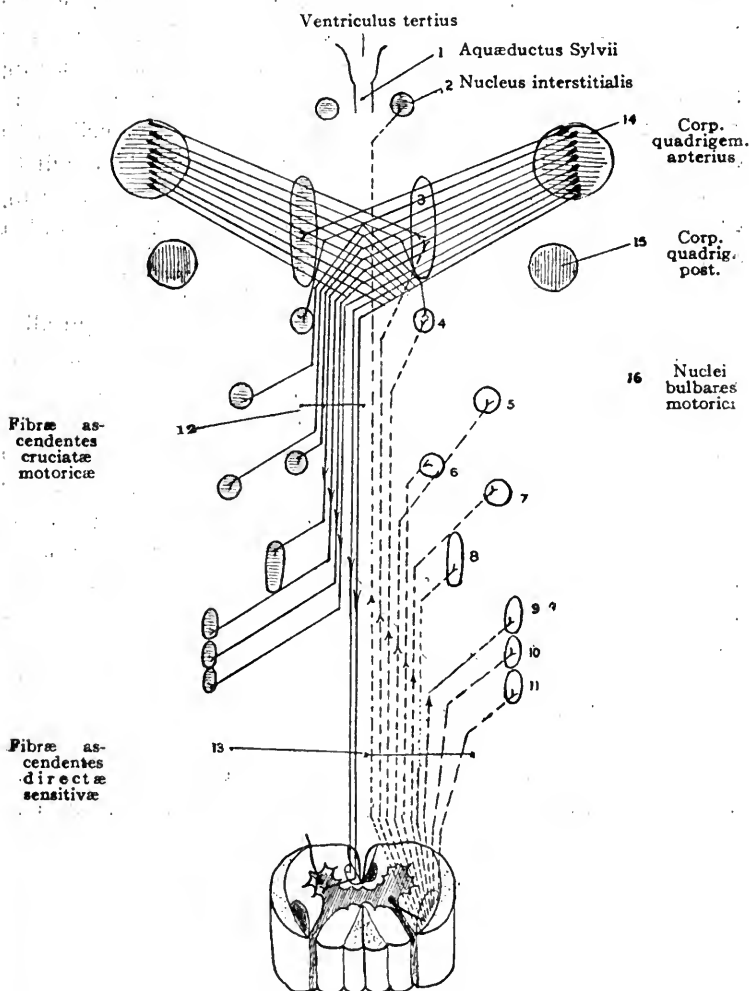


FIG. 8.—Sketch after Testut.

antero-lateralis to the motor nuclei of the cranial nerves. The homologous part of the trigeminus has been described by Mahaim as fasciculus latero-dorsalis, which goes from the termination of the trigeminus, *i.e.* from the superior part of

the columna gelatinosa, through the formatio reticularis in the fasciculus longitudinalis posterior.

(b) *Fibræ descendentes.*

These are the axis-cylinders of the ganglion cells of the corpora quadrigemina anteriora (according to Held) and the fibers of the ganglion cells of the nucleus interstitialis (according to Cajal); they conduct the excitation from the mesencephalic centers to the motor nuclei of the cranial nerves as well as to the anterior horns of the cervical spinal cord. It is well to bear in mind that in the dilatation of the pupil after lowering the illumination, we have two reflexes—a direct one, and a consensual one.

This may be illustrated by a sketch after Testut.

There doubtless exists an anatomical center for pupillo-dilatation in the cervical spinal cord. Attention was first called to it by Parfour du Petit (1727), who showed that section of the cervical sympathetic was followed by contraction of the pupil on the same side. Budge (1851-1855) was the first to give us precise knowledge regarding this center, which he called centrum cilio-spinale, by means of the following experiment: He found that if the spinal cord between the sixth cervical and the fourth thoracic segment was isolated by transverse sections above and below, stimulations of this area caused dilatation of both pupils, which disappeared on one side if the corresponding sympathetic was cut. Stimulation of the cord above or below this region caused no effect. Jacobsen localized this centrum cilio-spinale through anatomical examinations in a case of tumor in the ganglion cells at the apex of the lateral horns of the last cervical and first thoracic spinal nerve.

*(To be concluded.)*

BOLETIN DE LA SOCIEDAD DE OFTALMOLOGIA  
DE BUENOS AIRES, A. R.

(January, 1917. Vol. iv., No. 4,—435 pages. Buenos Aires, A. R.)

ABSTRACTED BY DR. M. URIBE-TRONCOSO, NEW YORK.

**H**OMAGE TO DR. P. LAGLEYZE. The first article of the book is a report of a special meeting of the Buenos Aires Ophthalmological Society in which Drs. Demaria and Arganaraz paid tribute to the memory of Prof. Lagleyze, considering him as the founder of modern ophthalmology in Argentina. He was Professor of Ophthalmology since 1889, and at the time of his death, which occurred in August, 1916, he had seen more than 200,000 patients with eye diseases in his clinic. Professor Lagleyze was a very accomplished surgeon and originated many operative methods, the principal ones being his operation for entropion, now used extensively in Egypt for trachoma, and his tucking operation of the eye muscles for strabismus.

His contributions to ophthalmology were very large. Many years before Von Hippel he described the aneurismatic degeneration of the retinal arteries. His work on albinism and a very valuable book on strabismus published in french are well known. Professor Lagleyze was the founder of the Buenos Aires Ophthalmological Society, now in its fourth year of existence.

DR. J. A. CABAUT. TWO CASES OF FILARIA LOA.

Report of two cases of this rare disease, in the first of which the author was able to remove the parasite, which was moving freely under the skin of the inferior lid, by means of a threaded needle passed through the skin around the vermes and tied quickly, in order to capture it. A small incision in the skin

allowed a forceps to catch and pull the parasite, which came out intact. It was six centimeters long by half a millimeter wide. Under the microscope it proved to be a *Filaria Loa*.

Another parasite was removed some days afterward from under the conjunctiva of the same eye. As it moved very quickly, Cabaut was only able to catch it with a forceps through the conjunctiva. In making the incision the vermes was cut in two parts and only the caudal end extracted.

Examination of the blood of this patient during daytime showed great quantities of embryos surrounded by a sheath. Marked eosinophilia.

In the second patient the parasite was also seen under the conjunctiva and the same method of extraction employed with partial success, due to the swiftness of the vermes.

Only six other cases are reported in the ophthalmological literature of Argentina; all in patients that, as those of Cabaut, have lived in Congo, Soudan, or other places in Africa, where it is very common.

DR. E. B. DEMARIA. HYDATID CYST INTO THE EYEBALL.

Echinococcus is very common in Argentina, but an endocular localization of this parasite had never been described. Demaria has been unable to find it in many hundred of enucleated eyes, sectioned at the clinic. This is the most surprising: 1st, because it is very common in all other parts of the body, even in the orbit (twenty cases reported in Argentina), and 2d, by comparison with the cysticercus, which has so marked a tendency to grow into the eye, so great indeed, that some writers have considered it as its habitual place of living.

In the whole ophthalmic literature there are only four cases reported of intraocular hydatid cysts, and of these only one is reliable. In fact both tenias, the solium and the echinococcus, live and migrate in the same manner in the body.

Dr. Demaria's patient presented slight pericorneal injection, iris completely applied to the posterior part of the cornea, lens cataractous, tension + 2; no light perception, projection lost. An intraocular tumor with secondary glaucoma was diagnosed, and a trephining operation done, which relieved tension for a few days; but a new attack of pain having set in the eye was enucleated.

Section of the globe gave way to a transparent liquid as



clear as "water from a rock" and a cyst was discovered, filling entirely the vitreous cavity and adhering to the retina and lens. The cystic membrane was characteristic. No daughter vesicles were present, but several proligerous vesicles, adherent to the wall and containing scolex, could be observed. An important characteristic feature was the absence of a pericystic membrane and of leucocytic infiltration, which the author attributes to the lack of inflammation of the tissues. The membrane is only a defensive reaction of nature against the parasite, and it seems that in this case the cyst acted only in a mechanical way during all its growth.

This lack of inflammation makes a most important difference with the cysticercus, which always produces a great irritation on the tissues of the eye: intense cyclitis, disorganization, and even sympathetic ophthalmia.

The author reviews afterwards in detail, the five cases reported in the literature (Gescheidt, Griffith, Werner, Wood, and Scholtz) and considers reliable only that of Werner.

The modern methods of sero-diagnosis (Appathie and Lorenz in Argentina, Guedini in Europe) have a great diagnostic value in echinococcus disease. It was positive in Demaria's case and is reliable and constant. Eosinophilia is to be found in about 67% of the patients and is not pathognomonic.

DR. E. B. DEMARIA. TWO CASES OF IRIDO-CYCLO-CHOROIDITIS, GRAVE AND BILATERAL, IN PATIENTS AFFECTED WITH VITILIGO.

In the first case, a young man 18 years old, the coincidence in the march of the ocular condition and the vitiligo was so regular that it could have been easy to correlate both conditions. In the second, a woman 45 years old, the uveal inflammation started six years after the beginning of the vitiligo, and was very severe; moreover a general examination did not show other pathological condition to which the ocular changes could be referred; still in the first case a tuberculin injection gave a strong positive general and local reaction, and in the second case Wassermann was positive. Demaria thinks that the vitiligo had nothing to do with the eye trouble, was a mere coincidence, and that the result of the tuberculin and Wassermann show the true nature of the disease. He even

doubts if in the cases reported by Gilbert in 1910 and Erdmann and Komoto in 1911, there was observed a true relationship between the vitiligo and the optic neuritis, uveitis, and chorioiditis, or if they were of other origin; because (except in the case of Erdmann) diagnostic tuberculin injections were not made. New clinical observations are therefore necessary to settle this point.

Demaria, however, thinks that a close relation exists between certain inflammations of the uveal tract and the condition of the pigment. He recalls the well-known heterochromia iridis of Fuchs, in which the lighter colored eye develops uveitis later in life, and the new theories on the causation of sympathetic ophthalmitis: the cyclo-toxin of Golowin and the anaphylactic theory of Elschnig.

DRS. DEMARIA AND CALDORA. ASSOCIATED MOVEMENTS OF THE SUPERIOR LID AND THE MASTICATION.

History of a case of this rare anomaly in which during rest a slight ptosis existed of the right eye, disappearing and even leaving exposed a part of the sclera above the cornea when the inferior maxilla was drawn downward in mastication. The elevation of the upper lid attained its maximum when the jaw was moved laterally in the opposite direction of the affected eye. The other ocular muscles were normal, except the pupil, which was larger than that of the other eye, although its reactions to light, accommodation, etc., were not impaired. The presence of this anisocoria is the principal interest of the case.

The anomaly is stationary. Sixty-eight cases have been reported to date, the first being that of Marcus Gunn in 1883. All these observations can be divided in three groups. In the first the elevation of the lid is produced when the mouth is opened and the jaw displaced laterally. In the second (less numerous) the levator palpebræ contracts only in opening the mouth, and in the third group only when the jaw is moved laterally.

The most general opinion of the cause of this condition is that the nucleus of the oculomotor is congenitally in relation with the trigeminus and even with the facial, the movements of mastication being principally commanded by the mesencephalic center which is near the origin of the oculomotor.

Lutz, however, opposes this view and holds that the disorder must be located in the subcortical centers. On the ground of the anisocoria observed in their case, the authors claim that the trouble must reside in the cortical centers, near the foot of the frontal convolutions, which are probably connected with each other by Meynert's U fibers.

DR. FERNANDEZ DE CASTRO. PARINAUD'S CONJUNCTIVITIS, TUBERCULOSIS OF THE CONJUNCTIVA.

History of a case of Parinaud's conjunctivitis with red and yellow granulations and ulcers in both lids, accompanied by swelling of the parotid and preauricular glands. The smear of the discharge and the inoculation of a piece of conjunctiva into the cellular tissue of the abdomen of a guinea-pig proved negative. Pus from the preauricular gland was sterile. However, histological examination of a piece of conjunctiva near one ulcer showed zones of necrosis and cells of Langhans, indicative of tubercular infection. Von Pirquet reaction was strongly positive.

A new inoculation of conjunctiva under the skin in a guinea-pig produced caseous glands in the groin.

The author considers his case as of undoubted tubercular origin.

DR. P. B. FERRO. CONTAGIOUS CONJUNCTIVITIS IN ARGENTINA AND ITS PROPHYLAXIS.

The author lays stress on the greater proportion of contagious conjunctivitis in Buenos Aires, which has increased from 9% in 1891 to 25% in 1917. This increase is especially due to a greater number of cases of trachoma, that has become prevalent all over the country. Altitude has no influence in its extension, as Chibret asserted. The increase is due to immigration of trachomatous patients, and the author insists on the necessity of the exclusion of these immigrants from Argentina, and the control by the Federal Government of all matters related to public health.

DR. R. GIL. PNEUMOCOCCUS CONJUNCTIVITIS.

Gil has examined bacteriologically in the clinic of Professor Demaria sixteen cases of acute catarrhal conjunctivitis of which nine (56%) were produced by the pneumococcus. This high percentage is in contrast with the statistics of European authors, who have found from 5% to 10% of pneumococcus

conjunctivitis, but agree with Gifford's figures in the United States.

DR. A. GOWLAND. VOLUNTARY NYSTAGMUS.

Before reporting his case, the author reviews all we know about nystagmus, its divisions, symptoms, and etiology. The involuntary or ordinary nystagmus is defined, according to Sauvigneau, "a convulsive phenomenon related to an irritative lesion of the center commanding the associated lateral movements.

Voluntary nystagmus is very rare and the cases reported in literature are very scarce. Dr. Gowland's patient was a man, 36 years old, in good health who only wanted to have glasses fitted. He attributed the diminution of his vision to a "voluntary twitching" of his eyes. He was able to produce at will a horizontal nystagmus, which persisted in every direction in which the eyes were moved. Pupillary reaction normal; no hippus, no anisocoria; fundus normal. Refraction: simple hypermetropic astigmatism of one diopter. During the oscillations all objects were seen moving. A strong concentration of will power upon the eyes was necessary to produce the condition. This voluntary twitching existed since childhood and was a source of merriment for him during his school days.

The father is dead; mother and two brothers living and in good health.

Voluntary nystagmus has been divided into three groups: rhythmic physiologic, true voluntary nystagmus, and the associated or Straunsky's, and the reflex or Bar's. Affergeld, who studied the first type, showed that it occurs with the lateral movements of the eyes, when rhythmic horizontal or rotatory, symmetrical oscillations of the globe supervene, especially marked on the side to which the eyes are rotated. It is observed in subjects without any disease of the eyes, ears, or nervous system, and is probably the result of the muscular strain in forced lateral movements. The extreme conjugate deviation of the eyes probably produces a stimulation similar to that provoked by vestibular stimulus, and extends to the rhythmic centers.

True voluntary nystagmus appears, according to Raehlmann, only in patients with marked visual anomalies which have existed from childhood, but Coppez's cases, and also the

one above described by Gowland, show that visual anomalies are not necessarily present. Nystagmography showed that contractions are irregular, without definite character, of variable duration, and changing from one twitch to the other.

Wecker has described a case associated with contraction of the pupil, and Coppez another with hippus. In this case the oscillations disappeared during the lateral movements.

The pathogeny of this rare condition, according to Coppez, must be ascribed to the following causes. In normal conditions the will acts only upon one center of association for moving the eyes in a lateral direction, but in nystagmus the voluntary stimulus goes to the centers on both sides, and if some kind of heterophoria is present the deviation at once appears. The position of the globes being modified and the ocular muscles subjected to an exaggerated tonus, react as usual by contraction, and nystagmus results.

The retraction of the lids during the twitching, proves that the levator palpebræ also receives an exaggerated stimulus.

Associated nystagmus, described by Straunsky in 1900, is a variety of voluntary nystagmus, due to the defensive reaction of the organism and produced in an indirect way. It occurs in patients suffering from palpebral and corneal lesions, or those with photophobia, when an attempt to open the lids is made. It has been observed also in perfectly normal subjects.

Bar described cases of conjunctivitis in which traction of the lid produced oscillations in the globes of an undulatory type, probably due to a reflex movement by stimulation of the trigeminus. This may be caused also by a foreign body, a corneal ulceration, or simply by any loss of epithelium of the cornea. The stimulus is transmitted to the bulbar nucleus of the trigeminus, and by the posterior longitudinal fasciculus to the nucleus of the oculomotor and its association centers.

DR. SANTOS FERNANDEZ (Cuba). PARALYSIS OF THE SEVENTH NERVE.

The author reports 70 cases of paralysis of the facial nerve observed among 50,000 patients in his practice, or about 0.11%, as compared with 0.76% for the paralysis of the other motor nerves. In these 70 cases the most important symptom

was: the mouth deviation in 44; the facial asymmetry in 15; lacrimation in 47; and lagophthalmos in 46; of the last 2 in leprosy patients.

The etiology was: "taking cold" in 52 cases; syphilis in 3; injury in 7; congenital in 3; with hemiplegia in 5.

DR. SANTOS FERNANDEZ (Cuba). RE-ESTABLISHMENT OF THE TRANSPARENCY IN OPAQUE CORNEAS.

IBID. THE OPERATION OF CONGENITAL AND TRAUMATIC SOFT CATARACTS.

DR. OTTO WERNICKE (Buenos Aires). TOTAL ACHROMATOPSIA.

Although partial blindness for colors is of common occurrence, total achromatopsia is very rare. The number of cases reported has increased recently, since we have learned to suspect the condition by other and more noticeable symptoms than the color tests.

Achromatopes learn to distinguish colors, and even to name them, by their respective difference in brightness, by the presence or absence of reflexes on the surface, by the form of the objects, and by habit. But for them the world is only an engraving with shades of gray and white.

They generally go to the oculist to be relieved of three important symptoms, viz.: low visual acuity, photophobia, and nystagmus.

Visual acuity generally varies from  $1/10$  to  $1/5$ ; rarely reaches  $1/3$ . Only one case is reported in the literature (Raehlmann) in which acuity of vision was normal.

Low vision is due in some cases to corneal scars, atrophy of the optic nerve, macular choroiditis, albinism, etc., but in the majority of cases there is no organic lesion which may account for it. The amblyopia is almost constant and not a mere coincidence, as has been suggested. This may give some clue for ascertaining the cause of the anomaly.

Photophobia is correlated to the third symptom: the nystagmus. When the former is severe the latter is very marked, and *vice versa*. Photophobia is a most striking symptom: the forehead is wrinkled, eyebrows contracted, palpebral aperture very narrow, head flexed. Dread of light increases with the intensity of illumination and decreases at night.

Luminous sense is normal in achromatopes. The nystagmus is rarely wanting, but it is not as constant as the amblyopia. Moreover in some cases it ceases with advancing age. It generally increases on fixation and is probably due to another very important symptom: central scotoma.

Roening was the first to detect central scotoma, which has been found afterwards in nearly half of the patients; but it is necessary to have always in mind that its detection is very difficult or sometimes impossible. Uththoff, who in one of his patients did not find it at first, was able to plot it a year later with a new method: the ring-shaped fixation test.

According to Grunert, even in cases in which central scotomata could not be detected, an anomaly of the macula or of the macular fibers certainly existed. This is the only way to explain how with very low central vision the limits of the visual field and peripheral vision in general were normal.

Achromatopes see red and the adjoining colors in the spectrum as a dark spot, and violet and near-by colors brighter than the normal eye. Red is mistaken for black. Yellow green is the brightest. The limits of the spectrum are either the same as normal or a shortening of the red end or of both ends may be present.

The best method for detecting achromatopsia is Holmgren's test. Hering disk and the spectroscope are also useful.

Dr. Wernicke's cases were two brothers, 18 and 12 years of age, born in a family of seven. No history of consanguinity. Daylight was troublesome and provoked continual blinking. If the elder brother kept his eyes open in strong light, after a few moments all appeared white to him. Vision improves at dusk. He is scarcely able to read and his school work was so deficient that he went to work in a hat store from which he was discharged because of his inability to distinguish colors.

Pupils react well to light and in accommodation. Fundus normal. Hyperopic astigmatism of half a diopter. Correction did not improve vision, which is only 1/10 for R. E. and 2/10 for L. E. Nystagmus is present and consists of small, very rapid, vertical movements, which do not increase on fixation. Red is mistaken for black and colors selected according to their brightness.

In the younger brother the symptoms were alike. Vision: R. E. 1/10, L. E. 1/10.

Clinical findings in cases of achromatopsia have exerted the greatest influence on the theories of color vision. Both Young-Helmholtz and Hering's theories needed to be modified in order to explain this condition. The first theory was modified by Parinaud and Charpentier and holds that the function of the cones is restricted to color sensations (black and white included), and that the function of the rods is the perception of light and dark and also black and white. These two systems do not work simultaneously. When the light is strong the cones transmit the sensations of all colors to the brain, but when illumination decreases, the cones cease to become stimulated, and then the rods begin to perceive sensations by the destruction of the visual purple. As the regeneration of this substance is very slow, peripheral vision is always bad in normal eyes, particularly in low illumination, for which the cones are not adapted. Hence the achromatopsia of the normal eye in darkness.

Total achromatopsia is due to lack of cones in the macula. This produces also the central scotomata and accounts for the poor vision of these patients and the nystagmus.

Hering's theory also is capable of explaining achromatopsia if modified to admit the duplicity of the function for the rods and cones as suggested by Parinaud and Charpentier.

It will explain perhaps more easily the case of Raehlmann in which achromatopsia was attended with normal vision and photophobia and nystagmus were absent.



REPORT OF THE PROCEEDINGS OF THE SECTION  
ON OPHTHALMOLOGY OF THE NEW YORK  
ACADEMY OF MEDICINE.

By LEWIS W. CRIGLER, M.D., SECRETARY.

GEORGE H. BELL, M.D., CHAIRMAN. MONDAY EVENING, DECEMBER 17, 1917.

Dr. ISAAC M. HELLER showed a case of **excessive secretion of the Meibomian glands**.

Dr. HELLER also presented a case of **xanthelasma that had been treated by fulguration with marked improvement**. He confined his treatment to one side only in order to study the effect of the treatment as compared with the non-treated side. The results were very apparent. The treatment was associated with moderate pain but there was no reaction or pain following the treatment.

Dr. JAMES G. DWYER read the paper of the evening: **Focal infections in the eye with special reference to the intestinal tract, with report of cases** (published in full in this number of the ARCHIVES).

DISCUSSION: Dr. JOHN E. WEEKS said that it was a fact widely known that toxins absorbed from the intestinal tract were responsible for a large percentage of eye disturbances and that he hoped that this line of attack would lead to more definite results.

Dr. E. S. THOMSON said that he had been interested in the relation between disturbances of the digestive tract and diseases of the eye for a long time. He said that heretofore eye troubles which he had traced to the intestinal tract as their underlying cause, would improve when treatment was directed to this cause, but recurrence was frequent. Dr. Thomson said that he was glad of the opportunity to test out the method

suggested by Dr. Dwyer. He said that he had had something over twenty cases under observation and treatment. Several cases of low-grade inflammation simulating glaucoma cleared up under the treatment. One case, in a woman 56 years of age, of iridocyclitis with light perception only, began to clear up in four or five days and recovery was more rapid than anticipated. One case with a corneal ulcer from fever, mucous colitis, and anemia got well in two weeks under the treatment. There were several cases of scleritis which seemed to improve as the intestinal condition improved; when the intestinal condition became bad again the scleritis would return.

Dr. L. A. COFFIN said that he had been interested in the relationship between the gastro-intestinal disturbances and focal infections ever since he began the practice of medicine, and that the longer he practiced the more he realized the preponderance of troubles springing from failure to correct the underlying cause—disturbed function and disease of the intestinal tract. The case to which Dr. Dwyer has asked him to refer was briefly as follows: A young married woman complained that she saw everything double. I took her immediately to an oculist, who gave her careful examination and told me which muscles were paralyzed. I asked, "What are the indications?" He replied, "bichloride of mercury." Again I asked, "What are the insinuations?" He replied that he thought I knew, to which I replied that I knew this lady's father, mother, husband, and son, and that I did not believe it. I then took the lady to the laboratory for a Wassermann blood test, the report being negative. The oculist told me that had the spinal fluid been examined, the report might have been different. The spinal fluid was examined by Dr. Dwyer. It too was negative. The lady was then put on generous doses of castor oil, intestinal irrigation, etc., and after about ten days' treatment, was entirely relieved of her diplopia from which she has not since suffered—now about two years.

Dr. B. W. KEY said that any focus of infection no matter where located is apt to excite a serous inflammation, and even destruction of delicately sensitive and actively functioning structures, such as the uvea and retina. He thinks Dr. Dwyer's studies and clinical reports a valuable contribution

to the already extensive investigation on the subject. In one case he had followed out Dr. Dwyer's line of treatment where it was found that the colon bacillus had almost entirely disappeared from the feces; there was one apical-tooth abscess however. The patient made a complete recovery without any further attack up to the present time. Dr. Key is unable to say whether or not the patient might have recovered under ordinary eliminative treatment with regulation of the bowels.

Dr. GEORGE H. BELL said that we should bear in mind that the gastric juice is not only a secretion but an excretion as well; that any thing that goes into the stomach which tends to produce more gastric juice than normal, tends to produce acid stools. He referred to the excessive use of cane sugar which breaks up in the stomach after fermentation into acids (two) and alcohols. He said that it was obvious in such cases that sugar should be eliminated from the diet.

By HENRY H. TYSON, M.D., SECRETARY *PRO TEM*.

MARTIN COHEN, M.D., CHAIRMAN. MONDAY EVENING, JANUARY 21, 1918.

Reading of the minutes of the meeting of December 17, 1917, by the Secretary *pro tem*. On motion, were approved as read.

Dr. JOHN E. WEEKS presented a case of **congenital coloboma of the macular region in both eyes** in a boy aged 5½ years. The colobomata were irregularly circular, having a diameter of about two millimeters in the right and three millimeters in the left eye. Vision R. 20/50, L. 1/200. The colobomata appear to affect the retina and inner layers of the choroid only. The retinal pigment was plentiful at the margins and there were a few small patches of retinal pigment scattered over the floor of the defects. The layer of large vessels in the choroid and the pigmented stroma of the choroid were intact. Retinal blood-vessels passed over a portion of the defect in each eye. The colobomata apparently have no relation to defects consequent on the imperfect closure of the retinal fissure, but are due to defects in the development of the choriocapillaris at the maculæ,—an explanation first advanced by Collins and Mayou.

Dr. ALFRED WIENER presented a case of **complete bilateral**

**ophthalmoplegia** in a man aged 67 years, following an attack of influenza. There was no loss of consciousness, and none of the other cranial nerves were involved. After six weeks there has been a gradual return of power in all of the muscles of both eyes, so that at the present time both eyes are practically normal. In view of the fact that this occurred in a patient with rather marked arteriosclerosis and came on suddenly, Dr. WIENER considers the ophthalmoplegia to be due to a thrombosis at the upper end of the basilar artery. He believes that one other explanation might account for this paralysis. A toxic agent circulating in the blood has been considered by some authors as a possible cause for abolishing function without visible changes. Such an opinion might be considered, but it hardly seems plausible.

DISCUSSION: Dr. LESZYNSKY thought that the possibility of polioencephalitis superior should be considered as a possible cause in this case.

Dr. WEEKS related a similar case examined by him about twelve years ago, coming on twelve days after the inception of grippe.

Dr. BLAKE referred to a case following influenza, which he thought was due to basilar meningitis.

Dr. ARNOLD KNAPP read a paper: **Return of tension after trephining, with a report of two cases.** The paper is published in full in this number of the ARCHIVES.

DISCUSSION: Dr. WILMER related a case in which at first an apparently perfect result was obtained, but later on account of return of tension, three separate trephinations and iridec-tomies were performed. Tension, previous to operations, between 30 and 40, reduced by trephining to about 14 would later rise to former high tension. Site of trephining would fill up with proliferated tissue. Case had tubercular history.

Dr. WEEKS stated that he also had had cases in which intra-ocular hemorrhages had occurred and trephining openings had been closed by iris, ciliary processes, and proliferated tissue. He has abandoned trephining except in very chronic glaucomas, buphthalmos, and like conditions. He also has had late infection. He thinks the operation is complicated by many possible mishaps, and he is now performing the LaGrange operation instead.

Dr. DENIG uses trephining in chronic simple glaucoma only.

Dr. GIFFORD considered trephining most suitable for chronic simple glaucoma and thinks it may be tried where tension is very high. He instructs the patient to use zinc collyrium daily during the rest of their lives in order to prevent late infection, and to pay particular attention to their conjunctival sacs. In buphthalmos his first two operations were unsuccessful on account of closure of trephine opening.

Dr. KNAPP asked Dr. WILMER for the cause of the proliferating process in his case. Dr. Wilmer thought that the tubercular lesion was the indirect cause.

Dr. RUDOLPH DENIG read a paper on **Transplantation of the mucous membrane of the mouth for various diseases and burns of the cornea, with a report of one hundred and fifty cases.** The first case was operated on by him in October, 1910. The results are most gratifying. Trachomatous and scrofulous pannus, herpes and allied processes of the cornea, lime and ammonia burns and burns caused by the contents of a golfball yielded quickly to transplantation.

DISCUSSION: Dr. WOOTTON was of the opinion that in some trachoma cases the Heistrath operation if performed properly gave good results, but that transplantation was a valuable addition as a last resort in old trachoma cases.

Dr. GIFFORD uses a Thiersch graft of mucous membrane from the lip which he obtains with a razor. He stated that pannus usually disappeared with the trachoma.

Dr. TYSON referred to a case of corneal burn with molten lead, in which he used a transplantation of mucous membrane, about four years ago, which still remains of a deep red color.

Dr. DENIG stated that if the transplanted flaps were too thick they remained red for some years, but eventually turned white.

REPORT ON THE PROGRESS OF OPHTHALMOLOGY  
FOR THE SECOND, THIRD, AND FOURTH  
QUARTERS, 1917.

By H. KOELLNER, Berlin; W. KRAUSS, Marburg; R. KÜMMELL, Erlangen;  
W. LOEHLEIN, Greifswald; H. MEYER, Brandenburg; W. NICOLAI,  
Berlin; H. PAGENSTECHER, Strassburg; K. WESSELY, Würzburg;  
and M. WOLFRUM, Leipsic, with the Assistance of Drs. ALLING, New  
Haven; CALDERARO, Rome; CAUSÉ, Mayence; CURRAN, Kansas City;  
DANIS, Brussels; GILBERT, Munich; GREENHOLM, Helsingfors; v.  
POPPEN, Petrograd; TREUTLER, Dresden; and VISSER, Amsterdam.

Edited by MATTHIAS LANCKTON FOSTER, M.D., F.A.C.S.,  
New Rochelle, N. Y.

I.—GENERAL OPHTHALMIC LITERATURE.

1. ADAMS P. H. The influence of vascular disease of the retina on the prognosis as regards life. *British Journal of Ophthalmology*, March, 1917.
2. CANTONNET, A. The usual alphabet for the blind. *Clinique Ophthalmologique*, vii., 2.
3. CHAPPÉ, TH. A few opportune operations. *Annales d'Oculistique*, cliv., 4.
4. CRUISE, R. R. Protection of the eye in warfare. *British Journal of Ophthalmology*.
5. DIANOUX. The abuse of enucleations. *Clinique Ophthalmologique*, viii., 2.
6. DOR, L. Late results after enucleation and evisceration. *Ibid.*, viii., 4.
7. DOR, L. Eyeglasses and masks. *Ibid.*, vii., 4.
8. GENET, L. Hemianopsias of cortical origin; evaluation of their compensation. *Annales d'Oculistique*, cliii., 4.
9. GENET, L. The blind of one eye. *Ibid.*, cliii., 6.
10. ROCHON-DUVIGNEAUD, A. The ophthalmological reform. *Ibid.*, cliv., 1.
11. ROURE. Recuperative ophthalmics. *Ibid.*, cliii., 4.
12. TEULIÈRES, M. The evaluation of loss of function in aphakic eyes of soldiers. *Clinique Ophthalmologique*, viii., 1.

ROCHON-DUVIGNEAUD (10, **Ophthalmological reform**) calls the attention of the profession to a number of reforms absolutely necessary to raise the French ophthalmology to a proper efficiency. He recommends the opening of a special eye hospital for children, a change in the methods of teaching of this subject, mostly for training ophthalmologists, the appointment of whole-time professors, the teaching of ocular physiology and physiological optics, which are not given anywhere in France, and the foundation of special laboratories for these branches of ophthalmology.

SCHOENBERG.

At the Ophthalmological Congress held at Oxford in 1917, Captain ADAMS (1, **The influence of vascular disease of the retina on the prognosis as regards life**) gave an account of an investigation which he had carried out to ascertain the fate of patients who had been found to suffer from vascular disease of the retina. The results of the research are so valuable that every medical man, whether ophthalmic surgeon, physician, or general practitioner, should make a careful study of the paper which expounds them.

The types of retinal lesion under consideration were, flame-shaped hemorrhages, venous embolism, retinitis circinata, and other conditions associated with arteriosclerosis.

Twenty years ago it was taught that persons who exhibited these retinal hemorrhages were liable to die within two years. Every observant physician soon became aware of the falsity of such a gloomy prognosis, and now Captain Adams has shown that under certain circumstances the prospect is really good.

He has collected 159 cases from the records of the Oxford Eye Hospital, and has traced 124 of them.

The critical factor with all these patients was soon seen to lie in the presence or absence of albumin.

Thus:

*With albumin.*

One died in five years of cerebral hemorrhage.

One died in five years of phthisis.

One died after three years ten months of Bright's disease.

One died in three years five months of nephritis.

One alive after two years three months.

One died in three months of granular kidney.

One died in four months.

One alive after six years.

*Without albumin.*

One alive after 26 years.

One alive after 25 years.

One alive after 17 years.

One alive after 16 years.

One alive after 11 years.

One alive after 4 years.

In many cases there was no note of the condition of the urine but there was ample evidence to state that when albumin is present the prognosis is bad and that when it is absent it is good.

A consideration of the cases grouped into age periods showed that the older the patient the better the prognosis, and *vice versa*, the younger the worse the prospect.

T. HARRISON BUTLER.

GENET (8, **Hemianopsias of cortical origin, evaluation of their compensation**) thinks that a practical way of determining the amount of compensation due to soldiers with cortical or subcortical injuries of the optic paths is to figure out the amount of loss of the field of vision. A normal eye has a field of vision of 10/10; both eyes 20/10. The loss of one eye is compensated for, by law, by 30% of the amount given for loss of both eyes. If 30% is awarded for a loss of 10/10 of the field vision, 3% ought to be awarded for a loss of 1/10 of field. On this basis it is easy to evaluate even charts showing an irregular outline of loss of field of vision. For instance: (1) A bilateral homonymous hemianopsia is, according to the author, equal to the loss of one eye. Compensation = 30%. (2) Quadrant hemianopsia. If a wounded soldier has lost a quadrant of 3/10 of the field in each eye he is entitled to 6/10 of 30% or  $6 \times 3 = 18\%$ . (3) If a wounded soldier has lost a portion of the field of vision exceeding 10/10 the compensation has to be figured somewhat differently. The first 10/10 of the lost field gets 30%, the value of the first eye. The remaining eye has a value of 70% ( $100 - 30$ ); consequently the remaining 10/10 are entitled to 70% compensation, and each 1/10 is to be awarded 7%. A concrete instance will illustrate



the subject. If a wounded man has lost  $7/10$  of the field in each eye, he is entitled to 30% for the first  $10/10$  of the lost field and  $4 \times 7\% = 28\%$  for the remaining  $4/10$  of the lost field. Total 58%. (4) Finally, a hemianopsia in an only remaining eye, the other having been enucleated, is to be compensated for in the same manner, as figured above. The first eye lost is compensated for by 30%. The  $5/10$  of the lost field of the second eye has to be awarded  $5/10$  of 70%, that is  $5 \times 7 = 35\%$ .

SCHOENBERG.

TEULIÈRES (12, **The evaluation of loss of function in aphakic eyes of soldiers**) estimates the partial loss of function and vision in aphakics higher than the partial loss of vision in non-aphakics. The aphakic eye, having lost two very important functions (binocular vision and accommodation), is entitled to more compensation than the non-aphakic eye with the same acuity of vision. He would award 5% for the loss of each of these functions. For cases of traumatic cataract of one eye in which both eyes, with correction, have useful vision, he would add 10% to the amount provided by law, as compensation for the loss of vision. Cases in which the non-aphakic eye had been blind or almost blind before the operation are entitled to less compensation, 5% above the indemnity provided by law, because they had no binocular vision before the injury. Cases in which both eyes have been operated on for traumatic cataract, with vision in only one, are, according to the author, entitled to 5% above the indemnity prescribed for the same reason as in the preceding cases. Finally those who have both eyes aphakic but with good vision in both are to be compensated by 15% added to the indemnity prescribed by law on account of their loss of both functions, accommodation, and binocular vision.

SCHOENBERG.

GENET (9, **The blind of one eye; statistics**) found that the proportion of one-eyed people, before the war broke out, was 0.81 per thousand. This fact he says should serve as a means of comparison for the future statistics of one-eyed individuals after the war. His material is composed only of men between 23 and 42 years of age who have had one eye partially or totally enucleated.

SCHOENBERG.

CANTONNET (2, **The usual alphabet for the blind**) says that

the only writing taught to the blind is the Braille alphabet, which takes a long time to be properly mastered. A large number of blind peasants and laborers never can learn this system of writing. The letters and figures of the author's new system consist of dots, in relief, arranged in such a manner as to imitate the outline of the ordinary alphabet and arabic ciphers. The advantage of the system is: (1) that it can be learned in from ten minutes to two hours; (2) that it can be read by seeing people as well as by blind ones; (3) the writing and the reading are done from left to the right (while in the Braille system the writing is done from right to the left and the reading from left to right). The Braille system has its own advantages, namely, it is read easier, it occupies less space, it is universally taught, and it gives the possibility of teaching music and stenography. For these reasons the author considers his own method as supplanting the older system. None of these two systems has all the advantages.

SCHOENBERG.

ROURE (II, **Recuperative ophthalmics**) refers to those cases of amblyopia in one eye from lack of use, traumatic cataract, or high astigmatism, which may recover a sufficient amount of vision when compelled to use these eyes. He advises to draft all the men with one such eye into the active army service because they can recover a good deal of vision in the amblyopic eye if their good eye should be destroyed by some war injury.

SCHOENBERG.

CHAPPÉ (3, **A few opportune operations**) says that by operating on soldiers with strabismus, pterygium, and corneal opacities, optical iridectomy in the last, a large number of them are made fit for active service. He advises to regard those who refuse an operation as malingerers who prefer the auxiliary service, and to be treated accordingly.

SCHOENBERG.

CRUISE (4, **Protection of the eye in warfare**) describes his visor of chain-mail which is arranged to hang from the helmet across the face. There can be no doubt that it affords adequate protection to the eyes with the minimum of disturbance of vision. The Reviewer, however, believes that he is correct in saying that it is not popular with the troops.

T. HARRISON BUTLER.

DOR (7, **Eyeglasses and masks**) has been told by the soldiers that they cannot wear eyeglasses under their masks on account of the blurring which results from the rapid condensation of vapor upon the glass. Consequently they have to remove the glasses every time they wear a mask. On account of this fact the author advises the military authorities not to enlist for active service myopes of more than 8 D. who could not find their way without glasses when compelled to wear a mask.

SCHOENBERG.

DIANOUX (5, **The abuse of enucleations**) protests against the readiness with which injured eyes are enucleated, many times without any real indication. He advises the utmost possible conservatism. In extreme cases he prefers evisceration on account of the better stump it offers.

SCHOENBERG.

DOR (6, **Late results after enucleation and evisceration**) has had occasion to examine sixty-three soldiers, twenty-three of whom had had one eye enucleated and forty had had an evisceration of one eye performed. He questioned each as to whether he continued to have any trouble in the remaining eye and obtained the following answers: Forty-one said that they had no trouble whatever; twenty-two complained of lacrimation, photophobia, periorbital pains, and accommodative asthenopia. Of these twenty-two patients twenty-one had had an evisceration performed, and Dor believes that this proves the superiority of enucleation.

SCHOENBERG.

## II.—RELATIONS OF OPHTHALMIC TO GENERAL DISEASES AND POISONS.

13. BALLANTYNE, A. J. **Quinine amaurosis with a report of a case.** *British Journal of Ophthalmology*, March, 1917.

14. BOURGUET and RONNEAUX. **Homonymous hemianopsia due to a serous meningitis localized in the right temporal fossa. Trepanation and cure.** *Annales d'Oculistique*, cliii., 7.

15. CALHOUN, F. P. **Antityphoid inoculations and ocular lesions.** *Ophthalmic Record*, November, 1917.

16. CHARLIN, CH. **Syphilitic meningitis with bilateral amaurosis.** *Annales d'Oculistique*, cliii., 11.

17. EVANS, J. J. **Fundus lesions in cases of war nephritis.** *British Journal of Ophthalmology*, August, 1917.

18. GABRIELIDES. Cysticercus of the brain accompanied by papill-  
œdema. *Annales d'Oculistique*, cliii., 5.
19. GLOAGEN. Herpes localized on the lids and cornea following anti-  
typhoid vaccination. *Ibid.*, cliv., 1.
20. GROSSMAN, M. The selective action of spirochetes. *Journal of*  
*the American Medical Association*, March 31, 1917.
21. LEVY, J. M. *et al.* Investigations as to the frequency of metastatic  
eye infections from primary dental foci. *Ibid.*, July 21, 1917.
22. MACKAY, G. A case of uveo-parotiditis. *British Journal of*  
*Ophthalmology*, October, 1917.
23. MORAX, V. Herpes of the mucous membranes and of the cornea  
following antityphoid vaccination. *Annales d'Oculistique*, cliii., 5.
24. MORAX, V. Observation of a case of palpebro-ocular herpes  
following a prophylactic antityphoid vaccination. *Ibid.*, cliii., 11.
25. MORAX and DUCAMP. A case of spontaneous arteriovenous  
aneurysm due to the rupture of an aneurysmatic internal carotid artery  
into the cavernous sinus. *Ibid.*, cliii., 6.
26. VAN SCHEVENSTEEN, A. Traumatic, incomplete, left homonymous  
hemianopsia, or quadrant anopsia, with conservation of the macular visual  
fields, accompanied by pure verbal cecity. *Ibid.*, cliii., 6.
27. WOOD, CASEY. Eye signs and symptoms of Mongolian idiocy.  
*Ophthalmic Record*, May, 1917.
28. ZENTMAYER, WILLIAM. The eye and the endocrine organs.  
*Journal of the American Medical Association*, July 7, 1917.

EVANS (17, **Fundus lesions in cases of war nephritis**) as the result of his experience as ophthalmic surgeon to the first and second Birmingham War Hospitals tells us that the lesions of the fundus met with in trench nephritis are slight in degree and are liable to be overlooked unless the pupil is dilated before the use of the direct method. He also is particular to state that he does not wish to imply that the lesions found are the result of the disease, they are only conditions of the fundus found in cases diagnosed as trench nephritis. Again no attempt has been made to differentiate types or degrees of nephritis. One hundred patients were examined: 43% had a normal fundus; changes were noted in the fundus in the remaining 57%.

The fundus changes observed involved the retina and the optic nerve in 39 cases, the choroid in 34 cases.

*Lesions of the Optic Nerve and the Retina.* These were slight in character, in fact in many cases doubtfully pathological. In 22 cases the disk was hyperæmic and slightly œdematous; in 13 there was peripapillary retinal œdema,

some perivascular lining, with tortuosity and fullness of the veins. In only one case was the classical appearance of albuminuric neuroretinitis seen. This man was obviously suffering from chronic Bright's disease. In no other case was any evidence of arteriosclerosis present.

*Lesions in the Choroid.* The author was surprised to find so many cases showing evidence of choroidal disturbance. Here again doubts could be entertained as to their pathological nature.

The chief varieties seen were disseminated choroiditis, and guttate changes at the macula similar to those seen in older subjects.

The most peculiar lesions noted were patches of slaty discoloration near the macula.

The process in both the retina and the choroid seems to be of a low inflammatory type. They seem to point to a toxic origin. The paper contains photographs of the organism.

T. HARRISON BUTLER.

ZENTMAYER (28, *The eye and the endocrine organs*) gives a complete summary of our present knowledge of the ductless glands, but the essay does not lend itself readily to review.

ALLING.

GABRIELIDES (18, *Cysticercus of the brain accompanied by papilloedema*) reports the observation of a girl of 17 with classical symptoms of a brain tumor and bilateral papilloedema. After trephining the skull subdural cysticerci were found, and the symptoms as well as the papilloedema were greatly relieved.

SCHOENBERG.

WOOD (27, *Eye signs and symptoms of Mongolian idiocy*) has had the opportunity to examine eleven cases of this condition. The ocular signs found are as follows: In two thirds of the cases the palpebral fissure was directed decidedly upward and outward, giving a "Chinese" appearance. Epicanthus was present in a quarter of the cases. Blepharitis and ectropion were also found in many. Convergent strabismus was seen in three and the common error of refraction was Hy. Astig. co. Nystagmus occurred in two instances. Cataract of the punctate variety is the most frequent ocular change. It is undoubtedly present quite early but is difficult to discover in infants as the dots are translucent. Changes in the

vitreous and fundus are not characteristic of the psychosis. The ocular defects are not of the familial type.

ALLING.

MORAX and DUCAMP (25, **A case of spontaneous arterio-venous aneurysm due to the rupture of an aneurysmatic internal carotid artery into the cavernous sinus**) report the following case. The patient, 68 years old, presented at the first examination a right-sided pulsating exophthalmus, continuous murmur heard over the temporal region, and paralysis of the third cranial nerve. He complained of an uninterrupted intracranial bruit. The symptoms appeared spontaneously during defecation three weeks previously. Subcutaneous injections of gelatine and ligature of the internal carotid artery were of no avail. The patient died about five weeks after the operation. A post-mortem examination revealed that an aneurysm had developed in the cavernous portion of the internal carotid. The rupture of the aneurysmatic dilatation into the cavernous sinus gave rise to the symptoms of pulsating exophthalmus. This rupture took place during a sudden increase of blood pressure produced by an effort during defecation. The patient had no symptoms before the aneurysmatic carotid had opened into the cavernous sinus. There are recorded in literature only two cases of aneurysm of the internal carotid in the region of the cavernous sinus, one by Dempsey and another by Coggin. Both had pulsating exophthalmus, and died some time after the ligature of the carotid, but the post mortem revealed no communication of the aneurysm with the sinus. In all of these cases extensive atheromatous changes of the cerebral blood-vessels were present which, according to the authors, explained the failure of the operative intervention.

SCHOENBERG.

GROSSMAN (20, **The selective action of spirochetes**) examined a family in which the father had spastic hemiplegia and pupils which did not react to light and only slightly to accommodation. The mother was also syphilitic. Three out of four children had paralysis of the sphincter pupillæ and ciliary muscle, with no reaction to miotics and some signs of congenital syphilis. Under treatment the father's eye did not improve, but in the case of the children there was partial

recovery. Ophthalmoplegia interna is a rare manifestation in syphilis. The lesion is presumably nuclear and must be due to a strain of spirochete possessing selective property for nerve tissue. This symptom may be the only evidence of congenital syphilis.

ALLING.

MACKAY, G. (22, **A case of uveo-parotiditis**) reports the following case. A girl, aged 30, was found to have slight iritis with complete ophthalmoplegia interna. No mydriatic had been used, and none could be traced. When admitted to hospital she developed bilateral swelling of the preauricular portions of the parotid glands. The highest temperature recorded was 99.4 F. The Wassermann reaction was negative. Tuberculin tests were also negative. No growth was obtained from the blood. There was a leucocytosis. A culture from the conjunctiva showed the presence of staphylococcus albus and citreus.

The glands did not become normal for six weeks. The cycloplegia slowly disappeared. Similar cases have been recorded by Heerfordt in which there was no known history of mumps.

Mackay thinks that the case for mumps as the sole cause of uveo-parotiditis is so far "not proven."

T. HARRISON BUTLER.

LEVY (21, **Investigations as to the frequency of metastatic eye infections from primary dental foci**), by treating the teeth of fifty-seven patients, has cured 14% of various inflammatory lesions of the eye and improved 37%. He is of the opinion that the infection, primarily of dental origin, travels to the eye through the lymph channels and not through the general circulation. He bases this on the fact that in all but one of the favorable cases the dental infection was on the same side as the affected eye.

ALLING.

CHARLIN (16, **Syphilitic meningitis with bilateral amaurosis**) reports about a patient 25 years old, with luetic changes in the nose, leucocytosis in the spinal fluid, and complete amaurosis in both eyes of four days' duration. The blindness was cleared up in the right eye by an intensive antiluetic treatment.

SCHOENBERG.

BOURGUET and RONNEAUX (14, **Homonymous hemianopsia due to a serous meningitis localized in the right temporal fossa**) report the following case. The patient, a prisoner of war, was struck by a shell in the right temporal region, and complained some few weeks later of headaches, loss of hearing, and noise in the right ear. The clarity of vision was normal. The field revealed a left lower quadrant homonymous hemianopsia. The diagnosis made was of serous meningitis, circumscribed in the right temporal fossa, pressing upon the right optic tract between the chiasm and the cuneus. A lesion of the occipital lobe was excluded by the absence of optic hallucinations. A trephining of the skull in the right temporal region was performed and from thirty to forty cc of fluid flowed out under pressure as soon as the dura was opened. Following the operation the hemianopsia receded gradually and at the end of about two months it disappeared entirely.

SCHOENBERG.

VAN SCHEVENSTEEN (26, **Traumatic, incomplete, left homonymous hemianopsia or quadrant anopsia, with conservation of the macular visual fields, accompanied by pure verbal cecity**) reports the history of a left-handed soldier, wounded in the right parieto-occipital region, who developed a left lower quadrant hemianopsia and pure word blindness. The four cases of quadrant hemianopsia reported by Marie and Chatelin did not show any aphasic symptoms. The author assumes that the lesion consisted of a destruction of the cerebral substance which extended deeply into the optic radiations and temporal lobe. The injury of the association fibers between the visuo-cortical center and the center of the mental representation of ideas has caused the partial alexia.

SCHOENBERG.

CALHOUN (15, **Antityphoid inoculations and ocular lesions**) has observed a case of acute iritis and one of choroiditis following antityphoid inoculations. One patient acknowledged previous gonorrheal infection and the other showed positive Wassermann. It is likely that the irritation of the injections served to light up a latent process. Similar complications have been observed in the French army.

ALLING.



GLOAGEN (19, **Herpes localized on the lids and cornea following antityphoid vaccination**) has seen three cases of this nature. The herpes appeared after the third injection in the first patient, after the fourth in the second, and after the first injection in the third patient. In each one the vision remained very much impaired on account of the central location of the corneal opacity.

SCHOENBERG.

MORAX (23, **Herpes of the mucous membranes and of the cornea following antityphoid vaccination**) reports a case in which the eruption appeared three days after the third injection and receded completely in one month.

SCHOENBERG.

MORAX (24, **Observation of a case of palpebro-ocular herpes following a prophylactic antityphoid vaccination**) reports a second case of the same nature. The patient set up a violent febrile reaction two hours after the third injection, and forty-eight hours later a herpetic eruption appeared on the lids of both eyes and the cornea of the right eye.

SCHOENBERG.

BALLANTYNE (13, **Quinine amaurosis with report of a case**) summarises his conclusions as follows:

(1) In quinine poisoning complete loss of vision may be found in association with a normal fundus, and there may be striking recovery of vision in spite of well-marked fundus changes.

(2) In all, or nearly all cases of quinine amaurosis, ophthalmoscopic changes, such as narrowing of the vessels, congestion of the disk, and opacity of the retina, make their appearance sooner or later, but there is no correspondence between the character or severity of these changes and the intensity of the visual defect.

(3) The visual defect cannot therefore be due to these changes, but rather to a condition of the retinal elements invisible with the ophthalmoscope.

(4) This condition may be induced or aggravated in the first place by ischæmia of the retina, but in the main it is caused by the direct toxic action of the drug upon the retinal elements. The ultimate recovery of central vision, with loss of peripheral vision and the failure of acuity in twilight, suggests that quinine has a selective action upon the rods.

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## III.—GENERAL PATHOLOGY AND TREATMENT.

29. SCARLETT. **The differentiation of varieties of diplobacilli by serological reactions.** *Annales d'Oculistique*, cliii., 11.

30. TERRIEN, F. **The X-ray treatment of visual disturbances due to the hypophysis.** *Archives d'ophtalmologie*, xxxv., 5.

SCARLETT (29, **The differentiation of varieties of diplobacilli by serological reactions**) has recently described two new species of diplobacillus which show specific characteristic cultural and staining properties. In this paper he reports a series of experiments which prove that the serum of rabbits, which have been repeatedly inoculated with bacillus duplex non-liquefaciens, contains specific agglutinins for this micro-organism but not for the diplobacillus of Morax or of Petit. This fact constitutes one more fundamental point of identification for the type of diplobacillus described by Scarlett.

SCHOENBERG.

According to TERRIEN (30, **The X-ray treatment of visual disturbances due to the hypophysis**), the surgical treatment of tumors of the hypophysis is successful only in exceptional cases. Since it has been established that the epitheliomatous, sarcomatous, and lymphomatous tumors respond very frequently to the X-ray treatment, several authors, Bèclère, Jeaugeas, Gramegna, have recommended and employed with satisfaction the X-ray in tumors of the hypophysis. Terrien publishes the histories of nine cases with tumors of the hypophysis from literature and one from his own practice which have been treated by X-ray, all with benefit. Headaches disappeared, vision improved, and the visual fields became larger. The rapidity with which the symptoms improve is remarkable. In seven cases the headaches ceased after the first treatment; the vision improved from 0 to 1/60-7/10. The results are so much the better the earlier the diagnosis is made and treatment instituted. The advanced condition of loss of visual function is no contraindication for the X-ray treatment. There are parts of the field, completely amaurotic, which recover good vision.

SCHOENBERG.

IV.—REMEDIES AND INSTRUMENTS.

31. COSSE. Ocular prothesis. *Annales d'Oculistique*, cliii., 7.
32. COULOMB and RUPPE. Oculopalpebral prothesis. *Ibid.*, cliii., 4.
33. CRIDLAND, B. The tonometer of Schioetz. *British Journal of Ophthalmology*, June, 1917.
34. DOR, L. A new universal optometric chart based upon the metric system. *Clinique Ophthalmologique*, viii., 1.
35. LLOYD, R. L. The stereoscopic campimeter slate. *Ophthalmic Record*, August, 1917.
36. STEIN, I. F. The use of sophol in the prevention of ophthalmia neonatorum. *Annals of Ophthalmology*, July, 1917.
37. VALOIS. Remarks on ocular prothesis. *Annales d'Oculistique*, cliv., 6.
38. VALOIS, G., and ROUVEIX. Prothesis for the one-eyed of the war. *Ibid.*, cliii., 12.
39. WOODS, H. Some phases of the diagnostic and therapeutic uses of tuberculin in uveitis. Illustrated by four cases. *Annals of Ophthalmology*, April, 1917.

Sophol is a silver methylene nucleinic-acid compound and contains 20% silver. STEIN (36, **Use of sophol in the prevention of ophthalmia neonatorum**) has used a 5% solution in 5257 cases of the new-born and has observed only two of ophthalmia neonatorum. In one of these the infection did not appear until the eighth day and probably was secondary.

These statistics are extremely favorable as compared with those during the use of nitrate of silver.

ALLING.

In three of WOOD's (39, **Diagnostic and therapeutic uses of tuberculin in uveitis**) cases a diagnostic injection of tuberculin had a focal reaction which resulted in permanent serious impairment of vision, due to opacities in the vitreous and acute choroiditis. He therefore warns against this danger in cases showing evidence of intraocular activity, but thinks that therapeutic doses should be given well under those required to produce focal reaction.

ALLING.

VALOIS and ROUVEIX (38, **Prothesis for the one-eyed of the war**) plead for more careful attention on the part of ophthalmologists to this subject. The authors have had to deal with a considerable number of injured soldiers who had their orbital contents more or less completely destroyed, and the introduc-

tion of an artificial eye was impossible. They describe their method of gradual distension of the small irregular space left after severe injuries of the orbit, or hasty enucleations, by the introduction of increasingly larger round or olive-shaped, well polished pieces of hard rubber. In order that the pressure should be exerted in a posterior direction they apply a compressive bandage during the entire course of treatment. The rapidity with which the space between the lids and orbital scar tissue is enlarged is surprising. Furthermore they have succeeded in deepening the conjunctival sac, and even in forming a new one, by the introduction of a dilator shaped like an eye speculum, which by its spring effect exerts a continuous mild pressure, upwards and downwards. Very frequently they used both types of dilators, the round and the speculum-shaped ones. Cases in which cicatricial bands prevent the introduction of any form of dilator have to have these bands carefully dissected out, and replaced by a Thiersch graft on which a dilator is immediately applied to keep it in place. The type of artificial eyes used by the authors differs entirely from that generally known. It consists of an anterior portion of hard rubber, painted to give the appearance of an eye, and a posterior portion of soft rubber, with air between these two parts. This kind of prosthesis has the advantage of being elastic posteriorly and responds more readily to the movements of the stump. The appearance is more natural and the unsightly depression above the upper lid, usually seen in patients with prosthesis, is not present in those wearing the new type of artificial eyes. (Unfortunately the paper does not describe how these artificial eyes are made, neither does it reproduce photographs of patients wearing these eyes.)

SCHOENBERG.

VALOIS (37, **Remarks on ocular prosthesis**) recommends the introduction of a piece of sclera of the enucleated eye between the lips of the conjunctiva in order to secure a more firm stump and greater mobility for the prosthesis. After each enucleation he advises the introduction of a piece of rubber, of olivary shape, behind the lids, which controls better the regular formation of the cavity in which the artificial eye is introduced later on. In cases with marked obstruction of the conjunctival sacs by cicatricial bands he uses dilators, made of rubber, of gradu-

ally increasing dimensions, which usually succeed in restoring the space necessary for the introduction of a prothesis.

SCHOENBERG.

COSSE (31, **Ocular prothesis**) gives his experience with 140 cases. It happened very frequently during these times of war that the stock of artificial eyes was exhausted and the author had to use a substitute, made of a material well known to dentists under the name of Crown Composition. It becomes soft when immersed in hot water, can easily be moulded, is well borne by the conjunctiva, and keeps its shape when cooled. The composition is softened by immersion in hot water and introduced into the conjunctival sac in such an amount as to fill up the entire space between the lids and the stump. The surplus is cut away with a knife. The mould is taken out and hardened by throwing it in cold water. On the anterior surface of the mould he paints the pupil, iris, and conjunctival blood-vessels. It often happens that the conjunctiva has been destroyed in part and replaced by cicatricial bands. The mould reproduces the exact configuration of the retropalpebral space and by making it a little larger at the point where the conjunctival sac is obliterated the continuous, gentle pressure restores a groove or makes it deeper, so that the prothesis can be better borne. The author has succeeded in restoring by this method conjunctival sacs which would have otherwise required extensive plastic operations.

SCHOENBERG.

COULOMB and RUPPE (32, **Oculo-palpebral prothesis**) have made an attempt to replace not only the eye but also the lids and the region surrounding the orbit. They found that of all the materials, metal, porcelain, celluloid, gelatine, etc., vulcanized rubber presents superior advantages, on account of its easy handling, color, and lightness. They reproduce a number of photographs of patients with partially or totally destroyed lids and orbital margins, showing how well they succeeded in constructing protheses which defied detection at a distance of even two meters. The authors have employed such means of restoration only in cases in which plastic work had been attempted and had failed, or in which the damage was so extensive that no surgeon wanted to undertake such work.

SCHOENBERG.

LLOYD (35, **The stereoscopic campimeter slate**) has devised a campimeter which combines the advantages of binocular vision as employed by Haitz, and Peter's method of placing the chart at a short distance from the eyes. A stereoscope is used which stands on a table and prevents the unsteadiness when held in the hand. The chart is a slate with crossed lines representing degrees, and covers a field of  $10^{\circ}$  up and down and  $25^{\circ}$  laterally. The instrument is used to examine the macular region and the blind spot. ALLING.

DOR (34, **A new universal optometric chart based upon the metric system**) gives a table of mathematical formulas by the aid of which charts consisting of circles superposed upon triangles of various sizes are to be drawn. These drawings could be clearly distinguished by normal eyes, at a distance of various meters, according to their size. The readings are in the metric system and the author has used this new chart, to the exclusion of Snellen's types, in a large number of cases with perfect satisfaction. SCHOENBERG.

CRIDLAND (33, **The tonometer of Schiötz**) has examined 1000 ordinary non-glaucomatous, hospital patients with the Schiötz tonometer with the object of determining the average reading. It was found to be 20.06.

The lowest record was 11.5, the highest 30.

A difference, averaging 2.43, was noted between the two eyes in 21.2% of the patients.

Cridland averages his results with other published figures and gets:

Normal reading 19.58 say 20.

Limits of normality—11.9 to 28.55.

He considers that any reading over 25 should be regarded with suspicion and indicative of glaucoma till further investigation disproves this diagnosis.

The use of the instrument and the precautions necessary to avoid fallacies are discussed.

The records are presented in a table which is cross classified into decades and state of refraction.

T. HARRISON BUTLER.

(To be continued.)

## ARCHIVES OF OPHTHALMOLOGY.

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### CONCERNING THE VALUE OF THE BLOOD COUNT IN THE DIAGNOSIS OF SYMPATHETIC OPHTHALMIA.

By S. R. GIFFORD, M.D., 1ST LIEUT., M.R.C.

SINCE Gradle, Ormonde, Browning, and Price-Jones emphasized an increase of lymphocytes in the blood of cases of sympathetic ophthalmia there has been some discussion as to the constancy with which this sign is present. In view of this disagreement, it seems that the report of careful differential counts on 9 cases of true sympathetic ophthalmia, 6 cases of recent perforating wounds of the globe, and 11 cases of prolonged or repeated non-traumatic inflammation of one eye, would not be without some value. It was intended to include repeated counts in a number of cases of chronic infection outside the eye, but circumstances have made this impossible, and it is hoped this data may be reported at a later time. The counts were made through the kindness of Doctor Harold Gifford on his private patients during the last two years.

*Literature.* Gradle (1 and 2) reported this increase in mononuclears in 6 cases of iridocyclitis after perforating injury, where there was danger of sympathetic ophthalmia. In one, the other eye had been affected and examination of the enucleated eye showed the changes of sympathetic ophthalmia. In the others, the condition of the second eye is not noted, so apparently no sympathetic ophthalmia developed. In all he found the mononuclears increased in some to over 40%. This

increase dropped to normal soon after enucleation of the injured eye. In 11 cases of perforating injury with quick healing, he found no such change, nor in 12 cases of non-traumatic iridocyclitis. The increase was most notable in the small lymphocytes. In a later report (3), he describes similar findings in 15 more cases of iridocyclitis after perforating wound. He mentions here finding cases of perforating wounds with uneventful healing which showed mononucleosis. He explains this change in both classes of cases according to Elschnig's anaphylactic theory, as due to absorption of uveal pigment sensitizing the organism to that pigment and producing the mononucleosis observed in sensitized animals. In the cases which show no inflammation, he assumes that there is slight absorption of antigen, but not enough to sensitize the organism, or else occurring in an organism not susceptible to such immunization.

Ormonde (4 and 5) and Browning and Price-Jones (6) at about the same time reported, in 9 cases of S. O. already developed, findings similar to Gradle's, except that the increase was most notable in the large mononuclears. They found that the count approached normal after salvarsan was given. Browning later (7) reports on 17 cases treated by salvarsan because of the similarity of the blood picture to that in protozoal diseases (1912). In 1913-14 (8) Browning again reports favorably on such treatment and notes that the typical blood picture develops before any signs of irritation in the second eye. But he also mentions 5 cases of S. O. with a normal count. He describes one which was normal for 3 to 4 weeks, counts being done twice a week, after which the second eye became inflamed and the mononuclears increased to 34%. He notes that other complicating conditions may lower the count, as in a case of pernicious anæmia and S. O. with normal differential count. He says his later counts, checked by Price-Jones, have not shown so high a mononucleosis, though some change is fairly constant.

Elschnig (9) had noted the occurrence of mononucleosis, but says it is simply one proof that the organism is reacting abnormally to the irritation of trauma or infection. The picture is varying after perforating wounds; either lymphocytes or polymorphs may dominate the picture, or it may be normal.



He says it cannot be maintained that mononucleosis is the specific reaction to the cause of sympathetic ophthalmia.

In the same way, Guilbert (10), speaking of the mononuclears in the exudate and in the tissues of enucleated cases, denies that this tells anything as to the cause of the uveitis; it merely shows a chronic inflammation. The more chronic the process, he finds, the more the lymphocytes preponderate.

Franke, in 1912 (11), reported counts in 51 cases of various kinds, made in an attempt to see if mononucleosis occurred in ocular conditions other than sympathetic ophthalmia. Of 21 old perforating wounds of the sclera followed by inflammation but quiet for some time when the count was made, 13 had 30% mononuclears and 2 more had 29%. Of 3 cases with some inflammation remaining, but not affecting the other eye, all had lymphocytosis. Six of ten perforating wounds of the cornea had lymphocytosis. Of 7 cases where enucleation had been done at first after perforating wound, he found mononucleosis in four. Two cases of contusion of the globe showed mononucleosis. He further describes a case of Mannhardt's and one of his own, both of whom showed a normal count some time after injury but required enucleation later because of inflammation of the second eye. He concludes that, since mononucleosis is present in so many cases where no S. O. occurs and absent in some where it does occur, its presence or absence is of no prognostic or diagnostic value in relation to sympathetic ophthalmia. He does not mention increase of the large mononuclears, but speaks of the mononucleosis as a lymphocytosis. He and Hack (12) refer to this condition in some further cases, and think it occurs especially in neurasthenic people.

Sattler (13) in discussing Franke's first paper says he has found the sign of value in diagnosing sympathetic ophthalmia.

E. A. Neumann (14) in a Berlin thesis, 1912, reports examinations in a large number of conditions. I was unable to find the original paper, but from reviews, it appears that these include 85 cases of recent injury and operative trauma with normal healing, suppuration after injury, traumatic iridocyclitis where S. O. was suspected; and tubercular infection, glaucoma, and non-perforating contusions. In each group, he

found lymphocytosis more or less frequently. He concludes that it has no prognostic or diagnostic value after injury.

From Fuchs's clinic A. Purtscher and E. Koller in 1912 (15) report results more in agreement with Gradle. In 9 cases of true sympathetic ophthalmia, 4 of which showed the characteristic changes at autopsy, lymphocytosis was found in all but one, averaging about 35%. The increase was mainly in lymphocytes the large mononuclears averaging 5%. In seven cases of traumatic iridocyclitis without S. O., the highest percentage of mononuclears was 27%, average 20%. They conclude that the sign is of diagnostic value. They mention that Wolfrum found an increase of lymphocytes in two cases of fresh S. O. and also note that Guilbert found this increase, but found no difference between the count in traumatic iridocyclitis and other cases of uveitis.

Of late the tendency in this country and in England seems to be towards acceptance of Gradle's or Browning's views. Thus Brownlie (16) advises blood counts at intervals between removal of a foreign body and recovery, to forestall S. O., but he gives no report of counts made. Pyle (17) also mentions it as important in diagnosis, but gives no particulars. Zentmayer (18), however, refers incidentally to a normal blood count in a case of beginning S. O. O. C. Hudson (19) reports the same in a case of developed S. O., the count being made by Browning. Treacher Collins (20) mentions one of his cases which had a normal blood count. Siegrist (21) found 40.5% mononuclears in a case, but six months later, after use of salvarsan when the eye was quiet, this count had risen to 47%.

#### METHOD OF STUDY

Where possible three white counts and differentials were made in each case, at intervals of 3 or more days. At least 200 cells were counted, unless otherwise noted. One cause of past disagreement has been the confusion between different observers as to what constitutes a large mononuclear. Gradle (2) has called attention to this, saying that the difference has been largely a personal matter and that the total mononucleosis is the important thing. Since I found a rather larger percentage of large mononuclears in nearly all counts than is considered

normal, it may be said that in this work any cell with a fairly clear, well-defined cytoplasm extending for more than two microns around the nucleus was considered a large mononuclear. Most were at least two to three times the size of a red cell, but some were included not much larger than a red cell, where the amount of clear cytoplasm seemed to put them plainly out of the class of lymphocytes.

Most of the cases were put on large doses of sodium salicylate, and it occurred to me that this drug might be a factor in producing a mononucleosis, as some other drugs, pilocarpine, etc., are known to do. Therefore in four perforating wound cases, counts were made before starting the drug, and at intervals after it was started. Four guinea pigs and four rabbits were given sodium salicylate both by mouth and by intravenous injection, the doses corresponding roughly to one grain per pound of their respective body weights, in a day. In two of the guinea pigs this was continued for 13 days, in two more for 6 days, in the rabbits for shorter periods. Counts were made before the drug was started, just after the first dose, at intervals of 2 or 3 days during its administration, and 3 days after it was stopped.

#### FINDINGS

*Effect of Sodium Salicylate on the Blood Count* of the 4 human cases (10, 11, 12, 13, included in Table B): two showed an increase in total mononuclears, one a slight increase followed by a drop to normal while the drug was being given, and one a distinct decrease. Realizing that the changing pathological conditions and the change from home to hospital life would render any such results of little value in judging the effect of the drug alone, it was hoped experiments on normal animals would be of more value. The blood picture in these was found to vary greatly and not constantly in any one direction. In both rabbits and guinea pigs the mononuclears always made up 40 to 80% of the total amount and their number bore no relation to the doses of sodium salicylate.

TABLE A.  
NINE CASES RECEIVED AFTER THE OUTBREAK OF SYMPATHETIC OPHTHALMIA.

HISTORY	DATE COUNTED	W. B. C.	L. M.	S. M.	TRANS.	TOTAL MONOS.
1. James — Seen 4 months after removal of R. E. for injury. L. E., vision at first shadows. L. E. shows iridocyclitis. Slowly became quiet under treatment with finally almost no light perception. No change while being counted.	3/14/18 3/24/18 3/28/18 Average	10,400 10,200 18,020 12,870	32.5 30.5 17.5 26.8	9 10.5 11 10.2	3 3.5 3.5 3.4	44.5 44.5 32 40.4
2. Alvin — Treated for S. O. 1 year before and now operated for seclusio pupillae. No stirring up of inflammation by operation. Vision at first shadows. Final vision not recorded.	3/10/18 3/17/18 3/19/18 Average	16,900 15,200 10,200 14,100	19 15.8 17 16.8	10.5 13.5 10 11.3	1 1.7 1.5 1.4	30.5 28.9 28.5 29.5
3. Mr. T — Seen two months after injury to L. E. R. E. inflamed for 2 days. Vision of R. E., 20/10, L. E. 20/200. Scarlet fever when first seen and counted. R. E. responded slowly and improved only when atophan was given. Finally vision 20/30, and quiet at last count. L. E. always quiet.	3/24/17 3/26/17 4/22/17 Average	17,800 24,800 8,016 16,872	18 23.5 26.5 33.3	8.5 4 16 9.5	.0 2.5 1 1.2	26.5 30 43.5 33.3
4. Mr. C — R. E. perforated by iron, May 11. June 10, L. E. flared up. Seen June 17, and R. E. enucleated. Long course, with many deposits on Descemet's membrane, but vision 20/20. Papillitis recurring when last seen. Still under treatment. Secs. of R. E. showed changes of S. O.	6/17/17 6/22/17 6/24/17 12/9/17 Average	10,440 7,940 6,440 5,600 7,605	15 27.5 32 23.5 24.6	2.5 18 22 6.5 12.2	15 2 3.5 2 2	18 47.5 57.5 32 38.8

HISTORY	DATE COUNTED	W. B. C.	L. M.	S. M.	TRANS.	TOTAL MONOS.
5. Susie L. — Had S. O. 8 or 9 years ago. Recurrence with influenza. Examination of globe shows changes of S. O.	5/6/16	not taken	23.5	1	7.6	32.1
6. Mr. H. — 5 weeks before seen struck by nail in R. E. Vision of R. E.: fingers at 6 ft.; L. E. V. 20/20. R. E. enucleated followed by improvement of symptoms at once. Counts 7 and 18 days after nucleation when eye was quiet with opacities on anterior lens capsule. Final vision 20/20 L. E.	12/22/17 12/23/17 Average	6,200 6,800 6,500	18.7 10 14.3	26.5 8 47.2	3.6 5 4.3	48.8 23 35.9
7. Mrs. L. — Seen December 20, 1915, 2 years after injury of L. E. R. E. shows acute iridocyclitis. L. E. blind. V. R. E. fingers at 2 ft. First count 3 days after arrival, while R. E. was suffering acute inflammation. At second count still irritated. Later rise of tension. Vision down to 2/200. Doubtful if true S. O.	12/23/15 1/3/16 Average	9,400 not re- corded 9,400	36.4 7 18.7	4.2 12.5 8.3	.4 .5 .4	35 20 27.4
8. J. — Seen September 14, 1917. R. E. hurt 6 months ago and blind. L. E. shows iridocyclitis and desemetitis. Vision L. E. 20/30. Enucleated R. E. Final vision L. E. 20/40. L. E. quiet when counts taken; sections of R. E. showed changes of S. O.	10/26/07 10/27/07 Average	7,800 7,990 7,895	37.5 30 33.7	13.5 19 16.2	3 3 3	54 52 53
9. John S. — L. E. injured by knife 15 yrs. before and R. E. began to fail 4 weeks after this. L. E. removed 5 yrs. before. Now vision of R. E., fingers at 1 foot. Seclasio pupillæ. Eye quiet. Seen only February 25, 1918.	2/25/18	6,900	9	9	2	20

## APPENDIX—TABLE A.

THREE CASES OF S. O. FOUND IN THE LITERATURE (IN CONNECTION WITH THE USE OF SALVARSAN) IN WHICH BLOOD COUNTS WERE RECORDED.

HISTORY	CASE OF	W. B. C.	L. M.	S. M.	TRANS.	TOTAL MONOS.
Perforating wound of one eye. Inflammation of second eye. Enucleation of first eye. Count before enucleation.	W. Zentmayer	14,000	6.8	8.4		15.2
Inflammation started in second eye after injury to first. Count by Dr. Browning, before salvarsan was given.	A. C. Hudson	not given	9	not given		
Case of true S. O. with repeated exacerbations of uveitis in second eye. 1st count before salvarsan. 2nd count after salvarsan, when eye was quiet.	Siegrist 1/27/12 6/21/12 Average	10,500 7,087 8,793	3 2 2.5	32.5 41 36.7	5 4 4.5	40.5 47 43.7

TABLE B.

SEVEN CASES OF PERFORATING INJURY WITHOUT INFLAMMATION OF SECOND EYE. ALL BUT ONE COUNTED SOON AFTER INJURY.

HISTORY	DATE	W. B. C.	L. M.	S. M.	TRANS.	TOTAL MONOS.
10. C— Injury of R. E. seen soon after on same day. Put on large doses of sodium salicylate.	3/18/17	8,720	9.5	17	3.5	30
	3/19/17	7,620	16	15	1	32
	3/22/17	6,660	21	19	5.5	45.5
	Average	7,666	15.5	17	3.3	35.8
11. K— 3/27/17. Perforating wound covered by flap and sodium salicylate started.	3/27/17	10,880	16	5.5	2.5	24
	3/29/17	9,532	19	11.5	1	31.5
	3/31/17	9,820	15	3	1	29
	4/1/17	5,900	12.5	8	4.5	25
	4/6/17	9,040	15.5	4	4.5	24
	Average	9,019	15.6	8.4	2.7	26.7
12. Mr. Y— 3/29/17. Piece of stick in right eye. Removed this day and sodium salicylate started.	3/29/17	10,350	7	7.5	3	17.5
	3/31/17	12,020	8.5	4	1.5	14
	4/2/17	7,560	22.5	12		36.5
	4/6/17	8,690	14.5	8.5	2	25
	4/18/17	6,640	10.5	31.3	4.6	46.4
	Average	9,042	12.6	12.6	2.4	27.6

TABLE B.—Continued

HISTORY	DATE	W. B. C.	L. M.	S. M.	TRANS.	TOTAL MONOS.
13. Mr. G— Cornea wounded by stick 2 days ago. Piece of stick removed and sodium salicylate started.	3/31/17	8,350	13.5	18	2	33.5
	4/2/17	13,020	17.5	8	5	30.5
	4/6/17	11,340	12	3	1.5	16.5
	Average	10,900	14.3	9.6	2.8	26.7
14. Donald C— Perforating wound of eye 3 weeks ago in child of 3. No inflammation of 2d eye. Injured eye showed some uveitis.	1/15/18	14,600	6.5	5.5	3.5	15.5
	1/29/18	20,140	16.5	8.5	3.5	28.5
	Average	15,370	11.5	7	3.5	22
15. B— Old perforation of cornea healed after some inflammation, under sodium salicylate with vision 20/15.	2/20/18	9,440	17.5	5	3	25.5
16. Joe C— Perforating wound of cornea R. E. Seen same day. No inflammation of second eye developed.	2/25/18	11,100	18.5	6.5	2.5	27.5



TABLE C  
ELEVEN CASES OF VARIOUS CHRONIC INFLAMMATORY CONDITIONS

HISTORY	DATE	W. B. C.	L. M.	S. M.	TRANS.	TOTAL MONOS.
17. C— Iritis for some months. Is 50 to 60 years old.	5/9/17	5,400	16.5	13	.5	30
18. K— Glaucoma with iritis for some months. Count made some days after paracentesis. Final enucleation showed changes of glaucoma, no changes of S. O.	6/25/17	8,480	27.5	10.5	3	41.1
19. M— Specific iritis treated by sodium salicylate some weeks before and after entrance.	3/7/17	14,860	11	15.5	5	31.5
20. M— 4/2/17. Old iridocyclitis. Getting 120 grains sodium salicylate a day.	4/2/17 4/16/17 4/18/17 Average	12,860 6,640 6,920 8,960	20.5 13.5 8.5 14.2	4.5 6.7 2 10.7	4.5 3 5 4.2	29.5 23.2 39.5 29.1
21. R— Old iridocyclitis getting sodium salicylate.	4/3/17	9,100	19	22	2.5	43.5

TABLE C—Continued

HISTORY	DATE	W. B. C.	L. M.	S. M.	TRANS.	TOTAL MONOS.
22. R— R. E. lost by accident 7 years ago. Old irritable stump remains. Now cataract of L. E. First count before any treatment. Enucleation of old stump. 2nd count 2 days later. Cataract operation on L. E. with good result, some weeks later.	12/7/17 12/11/17 1/7/18 Average	13,400 10,200 ? 11,800	14.1 14.5 15 14.5	10.5 8.5 11.5 10.2	1 .5 1 .8	25.5 23.5 7.5 25.5
23. A— Perforating wound of R. E. 2 months before. L. E. shows slight irritation. Vision about O. K. Referred to local doctor with advice to keep R. E. under watch. L. E. quiet.	1/7/18	6,400	18.5	8.5	2	29
24. B— Old wound with abscess of cornea. Count made after long stay in hospital on sodium salicylate with healing.	1/30/18	8,700	15.5	13.5	7	36
25. H— Had vernal catarrh this summer and for some years. Now quiet. No eosinophiles in secretion.	1/5/18	5,200	25.5	16	4.5	46
26. M— Old obstinate uveitis of unknown origin. Resistant to all treatment.	11/20/17 11/21/17 Average	7,200 7,000 7,100	18.5 15 16.7	8 16.5 12.5	5.5 2.5 4	32 34 33
27. Isador W— Old vernal catarrh. Eyes now look quiet but still itch.	11/23/17	7,000	17	24	.5	41.5

TABLE D  
NORMAL CASE SHOWING MONONUCLEOSIS

HISTORY	DATE	W. B. C.	L. M.	S. M.	TRANS.	TOTAL MONOS.
Author's own blood.	5/9/18	6,400	14.5	32.5	5	52
	5/11/18	7,340	32	19.5	3	54.5
	5/18/18	10,680	23	17	2.5	42.5
	Average	8,140	23.2	23	3.5	49.7

## SUMMARY

*Group A.* This includes 9 cases in which sympathetic ophthalmia had broken out in the second eye. The clinical diagnosis was made by H. Gifford, and in 3 cases sections of the enucleated eye showed the typical changes of sympathetic ophthalmia. In one case (Case 7) included in the table, the inflammation may not have been purely a sympathetic one. Of these, seven were counted during or shortly after the active inflammation. All of these, except the one doubtful case, had an increase in mononuclears above the normal. The highest count was 53%, the lowest 33.3%. All showed the increase, especially in the large mononuclears, the highest being 33.7% and the lowest 14.3%. On the two cases seen some time after the process had become perfectly quiet, one showed 29.5% of mononuclears (Ormonde calls 30% pathological) with an average of 16.8% large mononuclears. The other, on whom only one count was made, had only 20% mononuclears, 9% being large monos. In no case did the count change materially under treatment, or after enucleation, though in 5 cases the eye became fairly quiet, with good vision, before the last count was taken. Case 3 was complicated by scarlet fever, which caused much variation in the counts.

It was found generally that a leucocytosis of 12,000 or over made results variable and unreliable. In Group A, average total mononuclears was 34.9%; average large mononuclears 22.8%.

Of the three cases picked up from the literature, in which counts were made, 2 showed no increase in mononuclears which could be called pathological. The third had an average of 43.7% mononuclears, with only 2.5% large monos. and this mononucleosis was increased after the eye became quiet under salvarsan. In all Group A cases the inflammation had existed after the active process had set in in the second eye, and none earlier than two months after the injury, so that in all the process was a fairly old one.

*Group B.* In this group, on the other hand, acute processes are concerned. In all but two of the seven cases, counts were made within two days of the injury. In only one case did the average total mononuclear count exceed 30% (Case 10, 35.8%).

The lowest was 22%, the highest 35.8%. Large mononuclears were considerably increased in most, relative to the generally accepted normal. The highest was 18.5, the lowest 12.6, average 15.7. These figures refer to averages of several counts. In several individual counts, a mononucleosis, much above the normal, was found, as 45.5%, 46.4%, 36.5%, in three cases. If these had been taken alone and the count had been considered of importance in the fate of the injured eye, it might have been sacrificed unnecessarily, as all of these did well with no symptoms in the other eye. In only one of these was there any material decrease in mononuclears under treatment. Average total mononuclears, 25.9%.

*Group C.* Eleven cases of long standing affections of the eye, 6 of some form of uveitis, 2 of vernal catarrh, included because of the mononucleosis shown, one of old irritable stump left after a perforating wound which showed on microscopical examination no change suggesting S. O., one of long standing abscess of the cornea, one of suspected S. O., with very slight irritation of the second eye. It is notable that this last case was one of the few to show no marked mononucleosis (29%), though his large mononuclears were 18.5%. Only three of these cases showed an average of mononuclears under 30% and of these 3, two had 29%. The largest was 46%, lowest 25.5%, and average 35.1%. Four were over 40%. Large mononuclears were increased in all—highest 25.5%, lowest 14.2%, average 17.9%.

*Group D* includes only 3 counts made on myself within a period of ten days, and show that an apparently normal person may have a high mononucleosis. Average mononuclears 49.7%. Large mononuclears 23.2%. At other times counts on myself have been normal.

#### CONCLUSIONS

1. An increase of mononuclears was found to be fairly constantly present in cases of sympathetic ophthalmia. Average total mononuclears 34.9%.
2. This high count often persisted for a long time, even in perfectly quiet eyes, but in two cases was found reduced to normal after the eye had been quiet for years.
3. It was not found to be constant in fresh perforating

wounds which did not develop S. O., average 25.9%. It was sometimes found in these cases, however, in isolated counts.

4. A similar increase was found to be just as constant in other chronic affections, such as non-traumatic uveitis, abscess of the cornea, vernal catarrh, etc., as in S.O., average 35.1% (slightly higher than in Group A).

5. The increase in both cases was most marked in the large mononuclears, but the lymphocytes were also often increased. The personal factor must be considered here.

6. Large doses of sodium salicylate were not found to affect the count constantly in animals or men.

These results agree with Gradle and most of the above authors in finding the mononuclears increased in S. O. They disagree, however, with Gradle, Purtscher, and Koller, and with Browning in not finding the count change to normal immediately after enucleation, and in finding it persist in quiet eyes. One reason for this disagreement may lie in the fact that many of Gradle's cases had no actual inflammation of the second eye, and, as Elschmig says, might never have developed S. O. When the first eye was enucleated in these cases, the entire seat of inflammation was gone, whereas in the present series inflammation had already begun in the second eye, and persisted for a time after enucleation, some slight degree probably persisting even in eyes that appeared quiet. Browning, however, found the count to drop after enucleation even in cases with actual inflammation of the second eye. And it seems only natural that this should occur in many cases, though it did not appear notably in this series, when the source of inflammation in the first eye, whether of anaphylactic, protozoal, or infectious origin is removed. The more important point of disagreement between these findings and those of the above observers is in finding a similar count just as constant in cases of old uveitis and other chronic conditions. In this they agree with the larger series of comparative cases of Franke and Neumann and with Guilbert's experience.

7. It is the opinion of the author, therefore, that the mononucleosis is not specific for S. O., but is found as a reaction of the body to a chronic inflammatory process in the eye, of whatever cause. It is true, as Browning points out, that an increase in mononuclears occurs in protozoal and spirochætal dis-

eases, as malaria and syphilis, and, as Gradle says, that it occurs in sensitized animals and persons. But it is also true that, as any infection proceeds from the acute to the chronic stage, the primary leucocytosis, which principally involved the polymorphs, is replaced by a slighter leucocytosis, in which the proportion of mononuclears becomes increasingly greater. The mechanism by which this occurs is unknown, but apparently the mononuclears appear after exhaustion of the polymorph reaction has taken place. In view of our present uncertainty as to the course of S. O., this seems as reasonable an explanation of the mononucleosis which does occur in the disease, as any other.

In view, then, of the frequency with which it occurs in other conditions, and the occurrence of cases of true S. O. in which it is absent, it would seem very doubtful if the sign could be considered of any importance in determining the presence or absence of sympathetic ophthalmia, or the likelihood of its occurring in any case.

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## REPORT OF A CASE OF BILATERAL PAPILLŒDEMA DUE TO EMPYEMA OF SPHENOID AND ETH- MOID SINUSES. OPERATION AND RECOVERY.<sup>1</sup>

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THE patient, aged thirty-four, came to the writer's clinic at the New York Eye and Ear Infirmary for the first time on December 1, 1917, complaining of severe headache, which had forced him to give up his work two weeks before. He claimed the headaches were worse when he stood on his feet. He did not have the triad symptoms which generally accompany intracranial increase of pressure. There was no vomiting or vertigo. V. O. D. 20/20; V. O. S. 20/20. His blood pressure was: systolic 200, diastolic 110. His fields for form and color were normal except there was a slight enlargement of the blind spot. On examining the fundus of each eye I found that the patient had a marked case of papilloedema, with many hemorrhages. The optic disk in right eye could best be seen +9. Left eye could be seen best with +8, which is equal to about 3mm above the plane of choroid. The cedema and hyperemia were so great that the entire papilla, the physiologic cup, and the borders of the disk, including the scleral and pigment rings, had all disappeared and the whole picture presented the appearance of a solid mound. You could not see the vessels bending over the edge of the disks. They were lost in the excessive cedema of the surrounding retina. The engorgement manifested itself also in the enormous distention of the retinal veins. The arteries were smaller than normal. There were many hemorrhages on the swollen papilla and surrounding retina.

The patient was ordered into the Infirmary at once. Rest

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<sup>1</sup> Patient was shown at the Ophthalmological Section of New York Academy of Medicine on March 18, 1918.



in bed, milk diet, and magnesium sulphate in the mornings. His urine showed a moderate amount of albumin, a few hyaline and granular casts, and a specific gravity of 1020. Wassermann was negative. Examination of teeth and tonsils was also negative. Patient gave history of having had a cold for some time and having had a discharge from the nose.

X-ray plate showed cloudy ethmoids, cloudy sphenoids, cloudy frontals on left side, and cloudy antrum of Highmore on the right.

While he was in hospital getting a rest cure for his kidney condition, his vision declined to 20/70 O. U. On December 16th, Dr. S. L. Craig operated upon and drained his ethmoid and sphenoid sinuses for me. There was some improvement following this operation, but his headaches still continued and bothered him so he could not sleep.

On December 21st, I had Dr. Hunt, the neurologist, to see him, on account of his persistent headaches. Dr. Hunt thought, owing to the fundus lesion and the occipital headaches, that the man had, in all probability, a brain tumor, possibly in the region of the pons, but did not advise any operation at that time.

The patient was not doing well, so I got Dr. Craig to open up his ethmoids and sphenoids again, and also got him at this time to drain the antrum of Highmore. His temperature was 100.6°

This operation was done on December 29th. December 30th, patient still complained of severe pain in the head. By January 4th, patient felt more comfortable, and on the night of January 4th slept fairly well. The treatment of applications and irrigations for his sinuses was kept up. Ever since December 30th the patient had shown marked improvement. January 5th the patient left the hospital, against my wishes, as he said he felt so much better. He continued to come to the clinic, however.

On January 14, 1918, I showed this patient at the meeting of the New York Ophthalmological Society. None of the members present questioned the diagnosis, even at that time, as the swelling and œdema were still very high. There was some difference of opinion as to the ætiology; some of the members thought brain tumor the cause, others thought it was due to a kidney lesion, while some of the members agreed with me.

I have contended all along that it was a case in which the clinical picture suggested the action of some severe toxin circulating in the blood. Four of my reasons for that are: first, headache; second, high blood-pressure in a man aged thirty-four; third, involvement of the kidneys; fourth,

marked oedema of the retina and papilla, with many hemorrhages. There was evidently some irritating substance in the fluid that filled the intervaginal space of the optic nerve. It is well known that after fracture, and more particularly after caries and necrosis of the body of sphenoid, infective meningitis may occur and lead to thrombosis of the cavernous sinus. Then why could not an empyema of sphenoids and ethmoids give us this toxin in the blood, which produced the kidney disease and the vascular disease of blood-vessels of the brain? And as the skull is unyielding, increased intracranial pressure arises as a result of the transudation of lymph and serous effusion, so that the lymph of the brain flows off to the spinal cord and also forward into the intervaginal space of the optic nerves. Owing to the increase of intracranial pressure, the neurologist advised against a lumbar puncture.

February 15th, the patient has shown gradual improvement, the hemorrhages are absorbing, and the swelling and oedema of the optic nerve and retina are not so great, V. O. D. 20/30; V. O. S. 20/30. Blood pressure 170. Patient comes to the clinic regularly and receives treatment also for his sinuses. March 16, 1918, I had a chemical analysis made of his blood at Post-Graduate Hospital. Urea 21.2; uric acid 7.4; creatinin 2.0; sugar 0.149. His urea and uric acid are still a little high, but his creatinin content is normal. Dr. Killian assures me that his uric acid and urea will be normal soon, and he considers the man out of danger. His kidneys are normal now, according to the test made of his urine in the pathological laboratory of the Infirmary.

The more recent quantitative micro-chemical studies of the blood have indicated that among the non-protein nitrogenous constituents the estimation of creatinin is likely to be of special service from a diagnostic and prognostic standpoint. In other words, the accumulation of creatinin in the blood has shown itself to be a useful index of renal insufficiency. The handicap of high creatinin accumulation, the kidney is, apparently, never able to overcome. Myers and Fine, who have accumulated extensive statistics on this subject, put the range of normal creatinin from 1 to 2.5mg per 100cc of blood.

March 18, 1918, I showed this patient at the Section on Ophthalmology of the New York Academy of Medicine. Again there was some difference of opinion as to the cause of the trouble.

Blood pressure now systolic 150, diastolic 100. The patient now has normal vision, normal fields for form, and slight contraction for colors, with enlargement of blind spot. The patient has no scotomata. The hemorrhages in the

retina and the œdema in the nerves and retinae have been gradually absorbed. The surfaces of the disks appear untransparent and dense. The edges of the nerves are irregular and uneven, and the nerve heads look larger, showing a hyperplasia of connective tissue on the nerve heads and surrounding retinae. Also there are some white streaks along the veins and arteries near the disks. The fundus of each eye, considering the severe inflammatory affection, presents a beautiful picture and one that is seldom seen.

April 1st, the patient has returned to work and feels fine. Blood pressure, systolic 140, diastolic 85. Now what else could have brought about such a remarkable recovery unless it was some severe toxin circulating in the blood, produced by the empyema of ethmoid and sphenoid sinuses? His kidneys had no treatment while in the hospital, except for two weeks before the operation, then I had him on a milk diet, rest in bed, and magnesium sulphate in the mornings. With that treatment his headaches increased and his vision gradually declined. The rapidity with which the eyes and kidneys cleared up, the writer thinks, leaves no doubt as to the cause of the trouble. It only goes to show the importance of making a thorough investigation when the ætiology is obscure.

Birch-Hirschfeld recently reported three cases of optic neuritis due to ethmoiditis. Good results were obtained by treatment directed to an empyema of the ethmoids. This case of mine is all the more interesting as at least 80% of double choked disks are due to tumors of the brain, either large or small, and is highly significant of intracranial disease.

One word in regard to differential diagnosis of papillœdema. In brain tumor cases: (1) The papilla is swollen out into a mushroom-like protrusion so as to appear enlarged but still well defined; (2) the choked disk is double-sided and not combined with retinitis. Whereas in cases due to focal infection we have:

- I. More inflammation of the retina.
- II. More œdema of the retina.
- III. Generally there are many hemorrhages in the retina.
- IV. We have a papillitis of the choked-disk variety.

## THE INTIMATE RELATION BETWEEN THE EAR AND THE EYE AS SHOWN BY THE BÁRÁNY TESTS.

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IT was not until the last century that the knowledge of the relation between the ear and the eye became recognized. It is of interest to note that the very first work on the relation of the ear to nystagmus and to equilibrium was brought forth about the same time by two men who were working entirely independently. In 1825 Fleurens made excisions of portions of the labyrinths of animals and noted that it caused movement of the eyes and definite disturbances of equilibration. Perkinje at the same time conducted experiments in turning human beings and likewise observed the resulting nystagmus and vertigo. The efforts of all the earlier investigators were entirely along scientific lines; the practical application of these investigations fell to the lot of the Vienna school of otologists; in the past ten years, Robert Bárány especially has done his monumental work in extending the field of this study into the realm of neurology. Bárány received the Nobel prize of 1915 and he is to be regarded as the pioneer in the clinical application of this study of the ear and the central nervous system.

The purpose of this paper is to call to the attention of the ophthalmologist the governing power of the ear over eye-movements and equilibrium. It is not yet generally recognized that the ocular mechanism is dependent upon the ear stimuli for precision of movement. Steadiness of central fixation is made possible only by normally acting ears. Tonic impulses from the right ear continually tend to draw both eyes

to the left, and tonic impulses from the left ear continually tend to draw both eyes to the right. This is definitely proven in that a sudden loss of the function of the right ear invariably results in a rotation of the eyes to the right, because the tonic impulses tending to draw the eyes to the left are impaired, and there is a resulting spontaneous nystagmus to the left. This may be further demonstrated experimentally by the use of electricity. In applying the galvanic current to the right ear, by the use of the anode, which depresses its function, there results a drawing of both eyes to the right with a resulting nystagmus to the left. The kathode stimulating the right ear causes a drawing of both eyes to the left with a resulting nystagmus to the right. These diametrically opposite phenomena can be produced merely by reversal of the current. It is thus shown that ocular equilibrium in the same way as bodily equilibrium is dependent on normally functioning ears.

Aside from the production of nystagmus in this experimental way, the ear in many animals has a decided influence on ocular rotations. Barthels (*Klinisch Monatsblatt f. Augenheilkunde*, Sept., 1914) makes the statement that rabbits have no trace of voluntary fixation of the eyes, but that ocular movements depend entirely upon stimuli from the auditory apparatus, and that section of the acoustic nerves in rabbits produces complete loss of eye-movements. In extremely young children he says it is impossible to produce rotary nystagmus and, although the auditory apparatus is already exerting some influence, the results of ear-stimulation are irregular eye-movements. In those who have been totally blind for a long period of time, showing the peculiar searching movements which Barthels calls "nystagmus of the blind," these, he says, show much less reaction to aural stimulation than do normal individuals, and he believes that these purposeless movements, which have developed because the blind person is not aware of the position of his eye, act against the nystagmus induced by the aural stimulation, and he concludes from this that the nystagmus of the blind is entirely separate from that produced from the ear.

It is now definitely known that the ear consists of two organs of distinct and separate function—the cochlea which is the

organ of hearing and the vestibular labyrinth which is the sense-organ of balance. The equilibratory portion of the ear consists of two tiny sacs known as the utricle and saccule and of three semicircular canals; the utricle takes cognizance of movements in a linear direction antero-posteriorly, and the saccule of movement in a lateral direction; the semicircular canals are so constructed as to detect *rotary* movement of the body in all conceivable planes. The "horizontal" canal detects movement in a horizontal plane; the two vertical canals—the "superior" and "posterior"—always work together and detect movement in the sagittal, frontal, or intermediary vertical planes.

The new ear-tests consist of experimental stimulation of these semicircular canals. This may be done by revolving a person in a turning-chair, or by douching the ears with either cold or hot water, or by applying the galvanic current to the ears. Such ear-stimulation produces certain definite phenomena—a rhythmic nystagmus and a subjective sensation of turning—vertigo. The two and only two definite reactions from ear-stimulation are nystagmus and vertigo; because of this vertigo, the patient falls in a definite direction, and also when he attempts to find with his finger or his foot an object he has previously touched, he is unable to find it but "past-points" to the right or left, above or below, depending on direction of the vertigo. These various phenomena are invariably present in normal people, and furthermore always follow definite laws. These findings can be as definitely studied and recorded as can central vision or the field of vision. For example, turning the individual ten times to the right at a speed of two seconds to each turn, with the head in the upright position, stimulating both horizontal semicircular canals, produces a horizontal nystagmus to the left of twenty-eight seconds' duration, a subjective sensation of turning to the left, and "past-pointing" to the right. Douching the right ear with cold water 68°, head upright, stimulating the vertical canals, produces after forty seconds of douching a rotary nystagmus to the left, a sensation of falling to the left, "past-pointing" to the right, and falling to the right. Hot water produces exactly the opposite phenomena. Turning produces a mechanical movement of the endolymph

within the semicircular canal; douching the ears with cold water produces a chilling of the outer portion of the internal ear, causing a lowering of the specific gravity of the endolymph; it therefore drops and produces a circulation downward. Hot water causes the endolymph to flow in the exactly opposite direction—upward. Such is the complete control of the ear over the eye motion that a nystagmus of any type and in any direction may be produced “to order” by appropriate ear-stimulation. The eyes are always drawn in the direction and in the plane of the endolymph movement.

If stimulation of the semicircular canals causes the eyes to move, it is evident that there must be nerve-paths between the ear and the eye-muscles. Similarly a stimulation of the semicircular canal produces a conscious sensation which is called vertigo; it is evident that there must be a nerve-path between the ear and that portion of the cerebral cortex which receives this impulse. The recognition of the ear as the chief equilibratory organ is so recent that most of the intracranial pathways are still undetermined. A study of over 600 clinical cases, including a considerable number of operations and autopsies, in the department of Neuro-Otology at the University of Pennsylvania has brought out certain facts in regard to these pathways, some of them definite, others needing further analysis. Our present belief, in fact, is as follows:

1. The fibers from the horizontal semicircular canals pass through the VIII. nerve, enter the brain-stem at the junction of the medulla oblongata and pons, and continue directly to Deiters' nucleus and there divide into two pathways:

- (a) The vestibulo-ocular tract concerned in the production of the nystagmus. These fibers go from Deiters' nucleus to the posterior longitudinal bundle, through which they pass to the various eye-muscle nuclei, from which, through the III. and VI. nerves they are distributed to the eye-muscles themselves.

- (b) The vestibulo-cerebello-cerebral tracts responsible for the vertigo. From Deiters' nucleus this path enters the cerebellum through the inferior cerebellar peduncles to the three vestibular cerebellar nuclei of the same side, from which it proceeds upwards through the superior cerebellar peduncle and continues to the cerebral cortex from both sides, but more

particularly the opposite side, through the crura cerebri. The cortical areas which receive these fibers are postulated by Mills to be in the posterior portion of the second temporal convolutions adjacent to the cortical areas for hearing.

2. The fibers from the vertical semicircular canals have a very different course: after passing through the VIII. nerve they immediately ascend into the pons and at a point above the middle of the pons they have a division into two pathways similar to the division of the horizontal canal fibers at Deiters' nucleus.

(a) The vestibulo-ocular tract, the fibers entering the posterior longitudinal bundle to be distributed to the eye-muscle and finally to the eye-muscles themselves.

(b) The vestibulo-cerebellar-cerebral tract reaches the cerebellum through the *middle* cerebellar peduncle, entering the cerebellar nuclei of the same side; from this point the pathway is identical to that of the fibers from the horizontal canal, through the superior cerebellar peduncle to the cerebral cortex of both sides.

The internal ear and these intracranial pathways constitute our conception of the "vestibular apparatus," and a knowledge of this is of use to the ophthalmologist in the study of ocular palsy and spontaneous nystagmus.

From the standpoint of the clinician it is, of course, the neurological aspect of this recently acquired knowledge that is most valuable, and it is to the neurologist that the result of the tests of the condition of these tracts has been most useful. A greater stride has been made toward the accurate localization of cerebral lesions by the knowledge thus acquired than by any other one thing brought to the attention of medicine in many years.

The ophthalmologist is primarily concerned with the so-called "nystagmus tract," and has only a collateral interest in the pathways producing vertigo. Many cases of muscle paresis and paralysis have much of interest added to them by a study of the ocular motions produced by ear-stimulation, and at times information of great clinical value is obtained: for example, given a patient with loss of conjugate deviation to the left, should stimulation of the right ear draw both eyes to the left, it is proven that the tracts from the nuclei to



the end-organs are unbroken and the lesion is supranuclear.

No case of nystagmus can now be considered as completely studied unless the results of ear-stimulation have been noted, and this frequently throws much light on the subject, both as to the site of the irritation producing the nystagmus and as to the prognosis. Illustrative of this may be cited two cases seen by us not long ago in the Children's Hospital; both were brought in unconscious with rather vague histories, both apparently were somewhat similarly affected, each running a high temperature with constant purposeless nystagmic motions of the eyes. They were believed to be the results of intestinal intoxication. An eye-examination elicited nothing, the ocular structures being normal. Ear-stimulation with cold water in one case produced a normal response, the purposeless motion ceasing immediately and a rhythmic nystagmus taking its place; in the other case no change took place in the character of the ocular rotations after ear-stimulation. In the first case, an opinion was expressed that the child's cerebral structure had not seriously suffered, and from this it seemed that recovery was possible; in the second case, since the ear-stimulation did not pass through the pathways in sufficient strength to cause a normal response, it seemed that there had been much damage in the brain tissue, and that the child might not recover. Each prognosis was proved by the outcome to be correct.

In cases of muscle paresis it has occurred to us that ear-stimulation by means of electricity might be used therapeutically, as the alternate pull and relaxation of the changing current would possibly succeed in stimulating to action what nerve-fibers there were remaining intact and shorten the time of recovery. We have had no opportunity of trying this, so it is in the realm of theory, but we intend to work along this line in the near future.

## SOME PHASES OF THE VESTIBULAR NERVE PROBLEM.

- (1) DEVELOPMENT OF THE VESTIBULAR TRINITY. (2) FUNCTIONS OF THE CEREBELLUM. (3) THE PENDULUM  
MOVEMENTS OF THE EYEBALLS.

By JOHN DUNN, M.D., RICHMOND, VA.

THE author wishes to say that without study of Bárány's splendid contributions to the subject of vestibular nystagmus, the suggestions made in the following paper would have been impossible for him.

Sitting in a Bárány chair, with my fists balled on my knees and my head bowed forward until it rested on them I was turned five times to the right. (One of the well-known tests used in the examination of candidates for aviation.) In my efforts to sit up I felt a slight pull to the right at my right ankle joint. This pull was progressively greater at the knee, the hip and shoulder, and by the time my effort was at its height, I felt as though I were made of rubber and my body and head were bent violently to the right. This increasingly great pull from my ankle joint upward to my head was so distinct that it resulted in my asking myself the question, "Why should this be so?" Standing upright I now bent my head rather violently towards the right shoulder, and I found that I was reproducing exactly the body and head and extremity movements which I had experienced in my effort to sit up in the Bárány chair, only in reverse order. As in the former experiment, so in the latter, I could feel the efforts of fixation at the ankle, knee, and hip, while my body and head were making a still greater excursion.

Several thoughts at once suggested themselves. Why should the head fall through a greater arc than the shoulder, the shoulder than the body, the body than the hip, the hip than the knee, the knee than the ankle? Why was the whole body, including legs and arms, not drawn an equal distance to the right? Why to the right and not to the left? Why should ocular nystagmus form any part of this curious falling movement? Why should there be bound up together the vestibular triad, the body and extremity movements, the nystagmus or ocular movements, and the disturbed flow of the endolymph over the cristæ? To say that the latter does evoke the other two is no answer at all. It is merely stating a fact. Why should it do it? Why is it not equally possible to evoke from the eye movements of the endolymph and the body and extremities? Or from these latter the other two? These are fair questions, and upon their proper explanation is founded the understanding of the vestibular syndrome, so far as it contains only the above three elements.

"It would be important if the influence of head movements upon nystagmus should be exactly studied" (Bárány, *A. of O. R. and L.*, vol. xxi., No. 1, pp. 107-8).

When I stand erect and bend my head toward the right shoulder, what takes place every time I do so? What has taken place every time I have done so since earliest infancy? In the first place I bring into play the will. In the second place there is a contraction of the dextro-turning cervical muscles. In the third place there is a relaxation of the sinistro-turning cervical muscles. In the next place, in order that *the head may be accurately turned in exactly the willed degree*, there takes place a yielding to the right of not only the head, but of the body, of the shoulder and arm, and of the hip with the knee and ankle. To prove this beyond question all that is necessary is to stand erect and turn the head rather violently toward the right shoulder, fixing at the same time the attention on the changes in position which the body, shoulder, etc., make. The sensations at the various joints tell of their participation in the head movements. We experience at the same time a counter-balancing relaxation of the left side of the body, etc. This, however, is not all that takes place. Both eyes rotate (not turn) in the opposite direction, *i. e.*, to the left, and there

is a disturbance of the endolymph of the vertical semicircular canals. From earliest infancy through life these three things take place with varying degree of intensity every time the head is bent towards the right shoulder, and in reverse directions whenever the head is bent towards the left shoulder. There has thus come into existence the vestibular trinity, and thus too have come into existence those intimate and excessively accurate nervous connections which are necessary for the regulation and coördination of these three elements of the vestibular syndrome. The understanding of these simple facts make clear in a wonderful way the vestibular problem. We can see at once the necessity for body falling, for past-pointing, for directional nystagmus. We can see the necessity for rotary nystagmus and for horizontal nystagmus. We can see the necessity for special branches of the vestibular nerve to the vertical canals and of the special branch to the horizontal canals. If we hold our head in the so-called optimum erect position and turn it to the right, what takes place? The eyes move in the opposite direction, *i. e.*, to the left. There is an excess of movement in the endolymph, confined to the horizontal canals. There are necessary body and extremity movements and adjustment, similar in kind, although less in degree, to those aroused by the turning of the head toward the right shoulder. Turn the head to the left and a similar trinity is brought into existence but in the opposite direction. The individual contributions to this vestibular trinity come into existence all through life each time the head is revolved about the vertical axis. And so again come into existence the intimate nervous connections necessary for their coördinated continuance. It so happens that anatomically the peripheral nervous contributions at work exist in a small compass only for the vestibular nerves, hence it is possible by peripheral excitation to reproduce the other two elements of the trinity only in the case of the vestibular nerve. In considering the body and extremity movements, the ocular movements, and the directional disturbances of the endolymph, we must bear in mind that they vary greatly in the ever-changing calls for varying the position of the head. The three primary combinations, around which all the others center, are those which result from movement of the head about its three main axes.

How came it about that there is necessary for the proper correlation of these three vestibular elements so elaborately developed an organ as the cerebellum? Bárány (*loc. cit.*) says: "the chief task of the cerebellum is to tone up and increase the efficiency of the musculature." Exactly what these words are meant to convey I do not know. They are certainly too inexact for me to get from them any clear idea of the functions of the cerebellum. This organ has functions as definite as those of the cerebrum, and until we know accurately what these are we must continue to talk loosely of many features of the vestibular syndrome. As I see it, the cerebellum so regulates the muscular control of the spinal column and of the various joints of the extremities as to make possible the exact performance of any desired, *i. e.*, willed, movement or movements. For instance, with my right arm extended I will to pronate it. That this may be accurately done in the right direction and with the rapidity I may will a certain exact fixation of the neck, of the shoulder joint, of the elbow joint, of the wrist and finger joints has to be made, otherwise the willed movement of pronation is inaccurately done, over- or under-done. A proper understanding of this fact makes easy the interpretation of the various disturbances of the cerebellar function; hypermetria, asynergia, adiadokokinesia, tremor, disturbances of writing, atony, hypertonia, hemiparesis, past-pointing, etc. That there should be definite cerebellar centers with the functions ascribed to them by Bárány and in the positions claimed by him is entirely in accord with this view of the purpose of the cerebellum. There is no evidence that the cerebellum originates movements. Its centers respond to sensations reaching it from without. The nervous current, so to speak, has to be turned into its centers to arouse its responding activities. From what source or sources may these responding activities of the cerebellum be put into action? I will to raise my right arm. In this instance the source of the impulse is in the left cerebral cortex. I liberate impulses in the right arm cerebral cortical center. These impulses pass through the internal capsule. A part of them pass unbroken through the crus and the crusta of the pons into the anterior column of the opposite side of the cord and so on. Another part passes to the pontine nuclei. Why

to the pontine nuclei? And whence from there? These impulses on reaching the pontine nuclei are sent to the cerebellum. For what purpose? To adjust the spinal column, shoulder, and neck so that the arm can with exact precision be raised. This is as clearly as I am able to state the two elements of all-willed impulses from the cerebral cortex. One element, the direct, is to accomplish the definite willed movement or movements; the other, the indirect, enters the co-ordinating nuclei of the pontine region and are transmitted to the cerebellum where they originate impulses whose result is to adjust the body stem and the joints so that the willed movement can accurately take place; these impulses result in the primary cerebellar response, *i. e.*, the willed response of the cerebellum. There is, however, another source from which cerebellar activities can be aroused, and this represents the purely reflex side of the cerebellum. This source is all the sensory nerves which take notice of voluntary muscular activities, *i. e.*, the sensory nerve supply to the muscle sheaths, to the tendons, to the ligaments about the joints, to the fascia and to the skin. The prime object of these peripheral born cerebellar excitants is to maintain the result of the cerebral willed impulse until it is released and another cerebral born impulse takes its place. *This is the more important raison d'être of the cerebellum.* For instance, I will to take a book in my hand, I will to sit in an easy chair, to cross my legs, to get my body in a comfortable position, to hold the book at an agreeable distance from my eyes, etc. These acts having been accomplished, I read the desired length of time, interfering with the fixity of my comfortable position only to will to turn the pages of my book. The maintaining of my reading position is left entirely to the peripheral born cerebellar excitants which without further exercise of cerebral activities maintain reflexly and subconsciously the position of my body and extremities. It is out of place here to elaborate the details of this view of the functions of the cerebellum. It is, however, necessary to have it here clearly in mind. The cerebellum can originate no muscular movements in the sense in which the cerebrum can. The cerebellum has no tonic influence over the muscular system. The muscular atony, etc., which result from deprivation of cerebellar function

are purely manifestations of disuse of the parts concerned. The cerebellar functions just mentioned, *i. e.*, the primary or willed, and the guardian function, have charge of all of the voluntary muscular activities which originate in the exercise of the will.

Development of the vestibular trinity imposed upon the cerebellum a further intricacy of detail that exact correlation of the movements of the head, of the ocular muscles, and of the endolymph of the semicircular canals might exist. Under normal conditions the willed turning of the head about the vertical axis arouses those primary cerebellar impulses, whose object is to adjust the spinal column, shoulder joint, etc., that the turning of the head may accurately be done; it arouses the ocular extended vagus action so that both eyeballs turn evenly toward the opposite direction, and lastly it arouses motion in the endolymph of the horizontal canals and this to a greater degree in one than in the opposite canal. (This latter owing to the shape of the two extremities of the canals.) Of these three elements none are directly under the will. The adjustment of the body and extremities, the ocular movements, and the aroused vestibular sensations occur independently of, and are beyond, the control of the will, when once the machinery for the contraction of the neck muscles has been set in action; all of them are unwilled results of the willed movement of the head. That the unwilled coördination of these three elements may take place there have been developed connections between the cristæ of the horizontal canals and both the cerebellar cortical centers and the vagic ocular centers. When the head is turned toward either shoulder similar conditions are aroused except that in this case the movements of the eyes are rotary and that the endolymph of the vertical canals is brought into play. And similar nervous connections are made between the cristæ of the vertical canals and the cerebellum and the vagic ocular nuclei. Inasmuch as when the head is turned to either shoulder the trunk and extremities require a more elaborate adjustment of the muscles than they do when the head is turned from side to side, the central connections of the vertical cristæ are more elaborate than are those of the horizontal canals. This is proven by the wider head, body, and extremity movements

when the endolymph of the vertical canals are artificially stimulated than those that occur when the same degree of stimulation is applied to the endolymph of the horizontal canals. There is still a third axis about which the head can be moved. The head can be moved forwards and backwards. Pretty much all comment upon this fact has been omitted in the discussion of vestibular nystagmus. It is none the less of the first importance if we are to have a proper conception of the subject. When the head is moved backwards and forwards in the antero-posterior plane it is accomplished with far less call upon the trunk and extremity musculature than when it is turned in either of the other two planes. What is, however, of more importance is the fact that the muscular response is equal for the two sides. This is entirely different when the head is either turned laterally or toward either shoulder. Here, as we have stated above, the contraction of one side is accompanied by a relaxation of the other, and this relaxation is under the control of the cerebellar centers. Moreover, when the head is moved up and down, whatever disturbances of the endolymph take place they are equal for the two sides. Therefore, moving the head in the antero-posterior plane can result in neither past-pointing, nor in the production of nystagmus. Moreover, when the head is dropped forward both eyes move directly upwards, and when it is thrown backwards both eyes move directly downwards. The will cannot move both eyes at the same time directly upwards or downwards. This can be accomplished only by suppression of the willed control of the ocular muscles and by turning them over to the vagic control. (It is both of interest and importance to note in this connection that vertigo can be produced by moving the head directly upwards or downwards. This fact will be commented upon later.) In reading Bárány's paper (*loc. cit.*), I rightly or wrongly came to the conclusion that he teaches that the cerebellar cortical centers which he so accurately describes and locates all directly respond to vestibular peripheral irritation. My own idea is entirely different from this. I believe that only those cerebellar centers directly respond to vestibular peripheral irritation which have been developed along with and are of necessity a part of the vestibular trinity complex, *i. e.*, those centers whose hyper-excita-



tion produce body falling and past-pointing of the type made manifest by irritation of the cristæ. The cerebellar centers for pronation, supination, flexion, and extension, etc., are developed in connection with the primary and guardian cerebellar functions, as described above, and are directly associated with the cerebral motor cortical centers. This is a very important distinction and without its full appreciation no correct understanding of cerebellar function is possible. That tumors, etc., of the cerebellum disturb the functions of both the vestibular and primary cerebellar centers is easily appreciated when we bear in mind that their size is such that they may disturb both sets of fibers or centers.

What has been written above of the vestibular syndrome in relation to its genesis from movements of the head is further borne out by the fact that the facial muscles, the tongue, and the voluntary laryngeal muscles do not participate in it. In turning the head in its various directions, none of these muscles are brought into play, and so no inter-communicating cerebellar centers for their associate adjustment have been developed. There is another fact which has not received proper recognition in descriptions of the vestibular syndrome. This is the tendency of the head to over-fall in the direction of the slow movement of the nystagmus.

Let us consider the eye movements. An explanation of past-pointing and body falling has been given. An explanation of the details of the vestibular nystagmus is more complex. In the former case, we are dealing with a disturbance of movements in the domain of those parts of the brain in which are exercised the powers of the will: the cerebral hemispheres and cerebellar hemispheres for the extremity movements, the cerebral hemispheres and the vermis for the body movements. Of these two, the former belong to a later period in the development of the brain than the latter and this accounts for the position of the centers for movements of the trunk muscles. Around the vagi nuclei the whole of the brain in its future elaborate development arose. In man, the centers for the ocular movements are pushed, as it were, relatively far forward along the brain stem. This tells of two things: first, that the eye centers, as developed in the human family, are the latest addition to the brain stem; second, that room had

to be made for the connections with the fibers which regulate the willed movements of the eyes. The primal eye was a part of the vegetative system and responded to impulses from the vagus centers. In its elaboration by development it has remained both for its internal mechanism and its external muscular apparatus under control of the vagus system, although by its later connection with the cerebral cortex, it has yielded a share in the control of its rotation movements to the domain of the will. Thus it has come about that excitation of the vestibular terminals sends impulses to both the vegetative ocular nuclei and to those parts of these nuclei which have later developed as the result of the addition of a willed control over the external ocular apparatus. Thus again it has come about that the nystagmus has a double element. The one which, as in the case of nausea and vomiting, results from over-excitation of the primary stem centers and becomes manifest whenever this hyper-excitation takes place, provided the stem centers are still functioning. This explains "the deviation of the eyeballs in the direction of the slow movement even in deep unconsciousness," for here the willed control of the eye muscles has been removed, while the vagic control is still active. The second element of the nystagmus results from over-excitation of the centers which have developed as the result of willed control over the external ocular muscles. These are similar to the adjunct cerebellar centers for the willed movements of the body and extremities. These centers must be in the cerebellar cortex and are probably in the paleocerebellum. The manifestation of their hyper-excitation requires, as in the case of past-pointing and body falling, a call from the cerebral cortical centers. Their manifestation follows the same rules which determine the direction in past-pointing and body falling. To understand the double movement of the nystagmus, the slow and rapid, it is necessary to know what takes place with the eyes when the head is turned from side to side, up or down, or bent towards the right or left shoulder. Little has found its way into the textbooks about the pendulum movements of the eyes. These may be best demonstrated as follows: Hold a mirror before the face and while looking into the mirror turn the head from side to side. It will be seen that the eyes move in a direction opposite

and with a rapidity equal to the movement of the head. They maintain their parallelism, but their movements from side to side have an evenness, a lack of jerkiness, which is altogether absent when the head is held fixed and the eyes are turned from side to side. Their movement resembles that of a pendulum in its evenness. In this experiment the willed control of the eye muscles is entirely suppressed. There is no consciousness of this ocular movement. On the other hand, when the head is fixed and the eyes are turned from side to side, not only is there consciousness of the movement of the eyes, but there is a visibly demonstrable jerkiness of them. In the latter experiment we see the efforts of the eyes to respond to the will. The former gives the most complete demonstration we have of the extended vagus control over the external ocular muscles. The evenness of the pendulum ocular motions is something that cannot be obtained by the will when the excursions of the eyes are either extensive or rapid. Witness the tendency to nystagmoid movements when the eyes are turned by the will, *e. g.*, either to the extreme right or left; and, as just mentioned, the accompanying jerkiness of their movements, which are so great as to arouse within the brain consciousness of them. The pendulum movements belong to the period in the development of the eyes when they turned automatically to the light as such without references to interpretation of form. It is further to be noted that the pendulum movements, in whatever direction they occur and whether up or down, from side to side, or rotary, are opposite the movement of the head; while, on the other hand, the fixing movements which occur at the end of the pendulum movement is in the direction of the head movement. The former is an expression of extended vagus action; the latter of cerebral action. Both are brought into play with every movement of the head. Applying to the ocular movements the facts stated about the past-pointing of the extremities and body falling, we can see that the direction of the nystagmus is determined by the change in the position of the head and only secondarily by the change in directional flow of the endolymph in the semicircular canals. The slow movement of the nystagmus, to repeat, represents the response to irritation of the vagi nuclei and correspond to the pendulum motion.

The rapid movement of the nystagmus represents a past-pointing of the eyes similar to the past-pointing of an extremity and necessitates an element of will for its accomplishment. Or to express it another way, in turning the head rapidly from side to side there is suppression of the willed control of the external ocular muscles; while with the head fixed if we turn the eyes rapidly from side to side there is suppression of the vagic control.

It may not be out of place here to discuss one or two of the details of the vestibular trinity. When artificial disturbance of the endolymph of, say, the right horizontal semicircular canal has been induced, there appears, even though the head be kept from movement of any kind, a horizontal nystagmus. At first thought this fact would seem to overthrow all that has been said about the development of the vestibular trinity. There is no motion of the head and yet disturbance of the guardian impulses, *i. e.*, those originating in the cristæ are able to bring into play ocular nystagmus. Apparently all three elements of the trinity should be always brought into play when one of the others is. Why should this disturbance of the vestibular terminals to the crista in the right horizontal semicircular canal not also induce, even when the head is kept motionless, movements of the body or extremities, as well as ocular nystagmus? In the first place, the impulses from the excited crista find their way to the vagic ocular centers, which as we have tried to show are in no way under the control of the will and are disturbed by peripheral born impulses in just the same manner as are the other vagic nuclei whose over-excitation results in nausea or vomiting. Thus it comes about that the eyes are turned to one side or the other, dependent upon the nature of the excitant applied to the endolymph of the crista. This vagic turning of the eyes takes place, whether the eyes are open or shut. When the eyes are open the will makes an effort to overcome this vagic movement and nystagmus is the result. If the will turns the eyes in the direction of the vagic movement the nystagmic movements either cease or are markedly less apparent. As a further result of stimulation of the vestibular terminals to the crista of the right semicircular canal impulses are sent to the cerebellar centers, central and cortical. Of these latter, only

those receive an over-stimulation which have been developed as a part of the vestibular trinity, *i. e.*, those which are concerned in those regulations of the body stem and joints of the extremities which make accurately possible the willed movement of the head through the action of the neck muscles. Although this is briefly discussed elsewhere in this paper, it is here repeated because of its importance. I have shown that the ocular nuclei are so constructed that, although the microscope cannot demonstrate the details, it is possible to excite the vagic elements without exciting the cerebral elements. In the same way in the cerebellar cortical centers, as well as in the central nuclei, it is possible to bring into play the elements which have been developed as a part of the vestibular syndrome without exciting those parts which have been developed to make accurately possible the demand of the will, *i. e.*, the responses of the varying parts of the musculature to impulses from the various cortical centers.

Movements of the eyeballs occur without causing the sensation that external objects have at the same time a corresponding motion. This is true whether we consider the normal movements of the eyes or the uncontrollable nystagmic movements which occur from certain causes, *e. g.*, albinotic nystagmus and optic neuritic nystagmus. With vestibular nystagmus external objects have a movement.

The next question that comes up is, Can there exist, while the eyes are at rest, a constant subjective sensation that all objects in the field of vision are in motion? The two following cases are of interest.

Mrs. L., aged 38. Six years before she came under my care she suddenly had an attack of vertigo, so severe as to cause her to fall. Since that time she has never been free from vertigo, the intensity of which has varied greatly. At the time of her first attack she did not lose consciousness. In subsequent attacks she has done so "for a moment or two." In the ten days prior to her first visit to me there had been three of these major attacks. This is more frequent than is usual. The attacks are followed by the sensation of a chill. After the Bárány tests in which she fell and past-pointed normally, and in which the nystagmus appeared normal in direction, she was ill for three days, feeling all the time "the boat motion." It was two hours before

she could stand erect. Both ears respond equally in about fifteen seconds to a cold caloric test and the sensation of dizziness and vertigo, produced by this test, was intense, so great indeed that the patient cried piteously. She was much nauseated. There was no visible nystagmus produced by this test and no demonstrable past-pointing. It was *as though the cold produced its vertigo and nausea effect before the semicircular canals became affected*. She is always conscious of the right ear; as though there were "something deep down in it." Hearing, both ears, normal for all tests. Eustachian tubes patent. Drum membranes show nothing definitely pathological. When sitting erect, if she bends her head forward, ever so little, and looks forward and downward, the sensation aroused is as though the chair in which she is sitting and she herself are moving backward. When she lies down everything begins to whirl, she thinks worse when she lies on her right side. Eyes: vision normal. Fundi and media normal. Pupillary reactions normal. No exo-, eso-, or hyperphoria. No areflexa corneæ. She feels as if she wants to close the right eye; as though the lids want to drop. No visible loss of function of upper lid. No nystagmus, even for forced movements of eyes to right or left, up or down. No unsteadiness of eyeballs can be made out, either when a light spot in the cornea is watched under a magnifying lens, or when the optic disk is under observation by the direct method. Movements of the head to right or left, do not increase dizziness. She, however, is distinctly conscious all the time of her right eye. She feels as though she were "going blind in it." Color sense normal. Fields normal. The most interesting symptom, however, for us here is that everything in front of her is in motion. For instance, a pencil held vertically at the distance of three feet in front of her has a blurred outline and is never still. The pencil "moves from side to side like the pendulum of a clock," within, however, very narrow limits. This blurring of the outlines of all objects looked at causes a sense of indistinctness in everything, and makes her feel as though "she were going blind." General examination: Patient has worn for a considerable time a supporter for gastropexy. Has some enlargement of the thyroid; probably mild form of hyperthyroidism. Pulse 90. Patient was operated on several years ago for appendicitis, since which time her right inguinal region has never been entirely free from pain and she complains of many attacks of distention. Family physician says she has slightly retroverted uterus and some stenosis of cervix uteri. History of long-standing constipation.

Without going into the further history of the case there stand out several phases of it which interest us here. In the first place, the "neurasthenic" element is plainly visible, as demonstrated by the fact that the cold caloric test is equally over-active for both the right and left ears. In the second place, this test causes dizziness and nausea before any demonstrable effect is produced upon the fluid in the semicircular canals. Again turning the head from side to side does not increase the patient's symptoms, while bending it forward or backward increases them tremendously, so much so that the patient asked to be allowed to have her throat sprayed, instead of being required to gargle it, as the latter required her to throw her head backwards, while the former could be done while the head is in the upright position. The excited terminals are not those of the cristæ of the semicircular canals, for not all symptoms of their excitation are evoked by the various minor tests which so readily provoke dizziness and nausea. The absence of any deafness or noises in the ears, even in the ear of which the patient complains of a sense of fullness, shows that the cochlear branch is not involved. We are then forced to attribute the disturbance to our excitation of the third branches of the vestibular nerve, *i. e.*, the otolithic branches. This opens up the question whether the nausea we meet with in the various tests of function of the semicircular canals is to be attributed to over excitation of the cristal terminals or is caused by simultaneous involvement of the otolithic vestibular terminals. It seems not unlikely that the latter will prove to be the correct answer, and the same for the symptom vertigo. What is the cause of the "pendulum movement" of all objects looked at? Is it reflex excitation of the purely vagic element of the ocular nuclei? It further would indicate that the otolithic branches of the vestibular nerve belong to an earlier stage in the development of the nervous system than do the semicircular canal branches. It is a question further whether they have any direct connection with the cerebellar centers.

Mr. B., aged 42. Three weeks prior to examination had "a dizzy spell" with vomiting. Since that time the sensation of vertigo and unsteadiness of his body has, while gradually lessening in intensity, never left him. There

has been no return of the nausea. General examination reveals nothing of interest *save a patch of acute arteritis in the left optic disk*. Hearing: right ear, low and middle tuning fork not heard by air conduction. High fork heard well by air. Bone conduction normal for low and middle forks. Ringing in ear. Drum shows old deficiency in upper posterior quadrant. No discharge. Left ear normal. Eustachian tubes patent. Response to cold caloric normal. Patient is still "light-headed." If he turns his head suddenly, with eyes either open or shut, to one side or up and down or towards shoulders, vertigo immediately appears. Turning the eyes to extreme right or left produces a marked unsteadiness of the eyeballs, and in this movement there is visible a considerable amount of rotary nystagmus, as far as I could judge equal for a right or left turning of the eyes. Vision is normal. Pupils and media and external muscular balance normal. Everything in the visual field is "trembling like a mass of shaken jelly." There is no definite direction to the movement. This movement of all objects looked at produces a "sensation as though he was going blind." Attempts to fix small objects do not increase the movements of the objects looked at. Here we have a further demonstration of the fact commented upon, *viz.*: Of the nervous association of the lateral and up or down movements of the eyes with similar movements of the head. The absence of participation of the convergence movements of the eyes is a still further demonstration of the exactness of the nervous connection and control in the production of the vestibular syndrome. The more complete our knowledge of the elements of the vestibular syndrome becomes, the more clearly is it seen that each element is the result of the excitation of a definite set of nerve terminals, whose pathways and control connections are accurately made for the accomplishment of their several definite purposes. Only those ocular movements take part in the vestibular syndrome which are always, as shown above, brought into play with changes in the position of the head. The convergence movements and the iritic and ciliary movements, not necessarily participating in the movements of the head, do not appear as a part of the vestibular syndrome. This is repeated here as its full appreciation throws much light on the relation of the ocular movements and to disturbances of semicircular canal elements of the vestibular nerve. If a patient be seated in a dimly lighted room and a cold caloric test is done, in a certain proportion of cases the pupils will be seen to slightly contract. This pupillary response is not a part of the vestibular syndrome, but one of the manifestations of the pupillary skin reflexes. Again



in the case of Mr. B., when the eyes are directed forward, there is no visible demonstrable motion of the eyeballs. The eyes were carefully examined under a magnifying glass in several different ways. And yet everything seems to be in motion. The nature of this motion and of that in the case of Mrs. L., involved as they are in questions, which arise concerning vertigo are beyond the intended scope of this paper. Lastly Mr. B., although a phase of vertigo is constantly present when his eyes are open, has no sensation of nausea, not even when on rapidly turning his head the tendency to fall to one side appears. Compare in this connection the case of Mr. H., 65 years of age. Ever since he could remember the rapid throwing of his head backwards would produce vertigo—without inducing either nausea, vomiting, or the other so-called vestibular symptoms.

These two cases ask many questions. Foremost among them are, What are the functions of the utricle and of the saccule? Why has nature thought it best to subdivide the vestibular nerve so as to send some of its branches to the utricle and the vertical canals, and others to the saccule and the horizontal canal? In the order of development which has the priority, the vertical or the horizontal canals? Why should nausea be a sequence from disturbance of the ear? Why vertigo? (The latter seems to be easier to answer.) Do there pass centralwards from the cristæ, fibers some of which are destined to arouse stimuli in the oculomotor centers, others to assist through the cerebellar centers in stabilizing the head, spinal cords, and joints of the extremities for the fullest performance of willed movements of these parts? (If so, and the functional "tests of the canals" seem to indicate it then the cristæ are organs more delicately elaborate than has yet been understood.) Can nausea be induced by disturbance of the cristæ nerve end alone? Or is nausea a manifestation of disturbance of the otolithic branches of the saccule? (And utricle?) What are the effects upon the endolymph beyond the semicircular canals of disturbances of the endolymph within them? These and many others equally important unanswered questions show the need for a clearer understanding of the mechanical problems of the endolymph and of the anatomical structures of the labyrinth. Until the tide of the endolymph is fully understood the literature of the vestibular nerve will continue to be filled with guesses.

THE LIGHT PUPILLARY REFLEX, ITS PATH, AND  
ITS ABOLITION CALLED IMMOBILITY OF THE  
PUPIL TO THE LIGHT REFLEX, AND REPORT  
OF A CASE OF UNILATERAL ARGYLL-ROBERT-  
SON PUPIL, IN WHICH CONSensual REACTION  
EXISTED IN BOTH EYES.

By Dr. ANTON LUTZ, HAVANA.

(*Concluded.*)

(*With Figure 9 in the text, and Text-Plate XV.*)

3. Efferent path for pupillo-dilatation.

The fibers of the ganglion cells of the centrum ciliospinale leave the cord by the ventral roots of its corresponding nerves, enter the rami communicantes, and run through the ganglion stellatum and the ganglion cervicale inferius to the ganglion cervicale superius. Cl. Bernard (1858) showed that after the passage through the ganglion cervicale superius the truncus sympathicus cervicalis divides into two branches; one branch includes the vasomotor fibers which have no influence on the pupil, and which enter the plexus carotideus; the other branch includes the fibers for pupillo-dilatation, and goes through the cervico-Gasserian strand, independently of the plexus carotideus, to the ganglion Gasserii and passes thence into the first division of the trigeminus (nervus ophthalmicus), following the nasal branch which it leaves finally to enter the long ciliary nerves, thus avoiding the ciliary ganglion. The long ciliary nerves enter the eye on each side of the optical nerve, and running forward between the choroid and the sclerotic, pass through the ciliary body to be distributed to the iris. The experiment of Budge shows that after having cut the ophthalmic

branch (not the whole trigeminus), we can no longer produce dilatation of the pupil through excitation of the cervical sympathetic, and that the dilatation fibers therefore pass through the ganglion Gasserii in the nervus ophthalmicus. Braunstein (1894) extirpated the ciliary ganglion and produced mydriasis through excitation of the cervical sympathetic or of the nervuli ciliares longi. We must therefore conclude that the radix sympathica ganglii ciliaris consists in the vaso-contractor branch of the plexus carotideus. Angelucci Lodato found, after extirpation of the ganglion cervicale superius, degenerations in some of the large ganglion cells of the ciliary ganglion, as well as in ganglion cells scattered through the orbit. I think these ganglion cells must therefore be considered as intermediate stations of the vasomotor sympathetic. The experiments of Langley and Anderson (1892) with local applications of nicotine to the different sympathetic ganglia and injection in the blood show that the dilatation fibers run through the ganglion stellatum and ganglion cervicale inferius, and enter only in connection with the ganglion cells of the ganglion cervicale superius. Schultz (1898) showed that after extirpation of the ganglion cervicale superius, it was yet possible to excite the musculus dilatator iridis and that it needed some days before the degeneration of the post ganglion fibers reached the musculus dilatator pupillæ.

The terminal organ of the pupillo-dilatation path is the musculus dilatator iridis, the existence of which the physiological experiments of Langley and Anderson made probable in 1892. They introduced electrodes through a wound in the limbus corneæ, and produced electrically a radial contraction of the iris. Later (1898) Karl Grunert proved the real existence of the musculus dilatator iridis through splendid anatomical investigations.

Considering these facts we have the following

*Chain of Neurons in the Pupillo-Dilatation Path.*

1st. Unknown path of the different sympathetic fibers of the cranial nerves (fasciculus longitudinalis post.) through the mesencephalon and bulbus medullæ.

2d. Ganglion cells of the centrum ciliospinale, whose fibers

## SCHEME OF THE PUPILLARY LIGHT REFLEX-PATHS

The scheme is incomplete in so far as it only shows the basal reflex-path. In order to avoid a too complicated design there are omitted: (1) the vasomotor reflex arc, which goes through the ciliary ganglion; (2) the cortical reflex arc, which begins in the corpus geniculatum externum, goes up and back through the radiation of Gratiolet to the calcarine fissure, where it finds its reflex arc in the stripe of Genari, and returning the same way descends to the pulvinar. The last step of it goes probably through the commissura cerebri posterior and is drawn in the scheme. Its existence is physiologically proven in so far as an electric excitation of the calcarine cortex produces marked miosis.

The basal reflex-paths consist of:

### A. SENSORY AFFERENT PATH.

- (1) Cones and rods.
- (2) Multistratified bipolar cells.
- (3) Multistratified ganglion cells.

N. opticus, chiasm, tractus opticus, brachium conjunctivum.

Affected in:

- (a) Amaurotic light immobility.
- (b) No suppression of the consensual reaction.
- (c) Hemiakinesis with hemianopsia.
- (d) Hemiakinesis without hemianopsia.

### B. PUPILLO-MOTOR CENTER.

(Deeper layers of the corp. quadrig. ant.)

### C. EFFERENT PUPILLO-CONTRACTION PATH.

- (4) Fasciculus tecto-bulbaris (origin in corp. quadr. ant.).
- (5) Fibers of the oculomotorius (nucleus photomotoricus).
- (6) Nervus ciliaris brevis (origin in ganglion ciliare).
- (7) Muscle sphincter iridis.

Affected in:

- (e) Pure direct unilateral Argyll (with consensual reaction in both eyes).
- (f) Total unilateral Argyll.
- (g) Iridoplegia with medium mydriasis.
- (h) Iridoplegia with marked mydriasis.

### D. PUPILLO-FIXATION PATH.

#### I. Active efferent path:

- (8) Fasciculus tecto-spinalis (origin in corp. quadr. ant.).  
Descendent fibers of the fasciculus longitudinalis post (origin in the sensitive cranial nerves).  
Spinal reflex arc of the sensitive branchial nerves.
- (9) Nervus sympathic. cervicalis (origin in centrum ciliospinale).
- (10) Nerv. sympathic. cranio-basalis (origin in ganglion cervicale supremum).
- (11) Musc. dilatator iridis.

#### II. Passive afferent inhibition path:

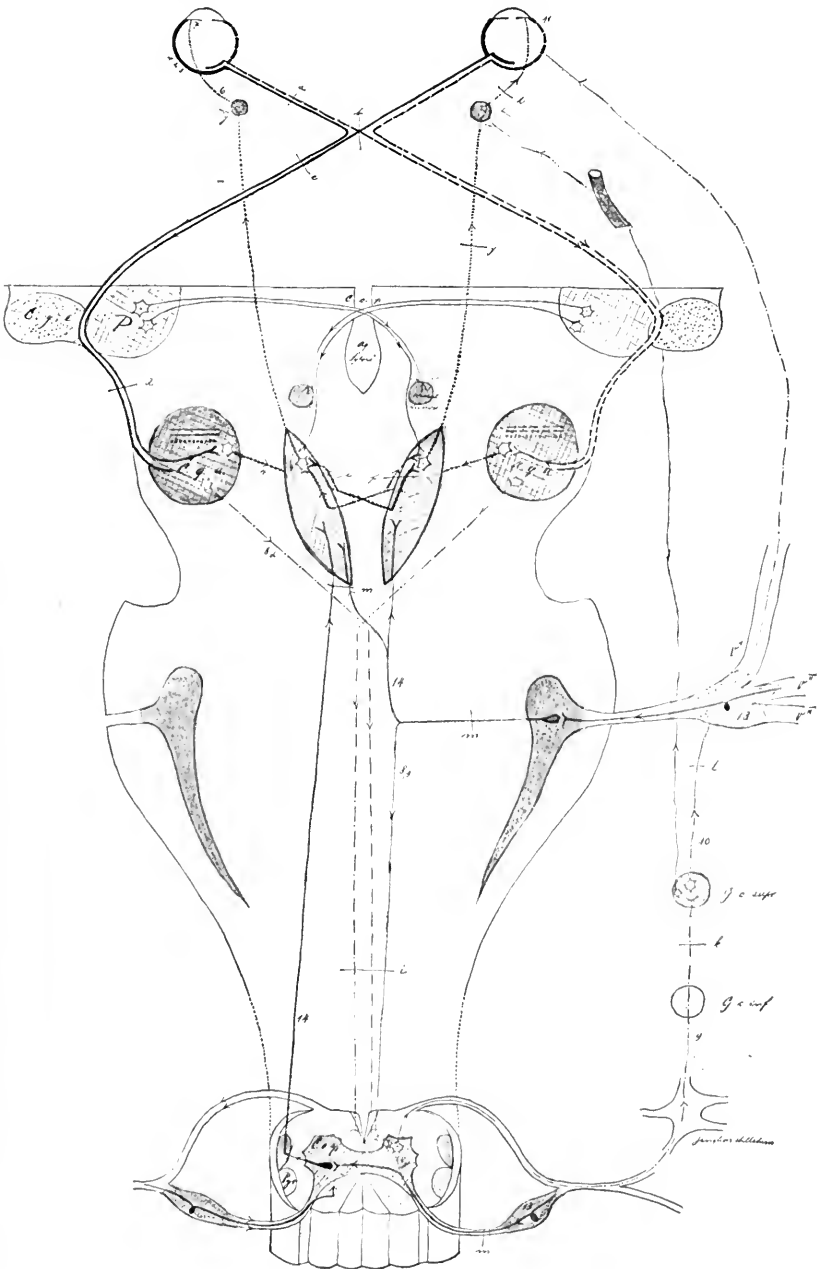
- (12) Sensitive end organs in the skin.
- (13) Ganglia spinalia, Gasserii and their homologues.
- (14) Ascendent fibers of the fasciculus longit. posterior.  
" " " " " latero-dorsalis Mahaimii.

Affected in:

- (i) Bulbar miosis.
- (k) Spinal miosis.
- (l) Hommer's miosis.
- (m) Abolition of the pupillary dilatation through pain sensation.

Also the basal reflex-path is not completely drawn. The spinal afferent pupillo-dilatation path only shows the path for pain sensation; the path of the other sensory ways (touch, etc.) which go through the nuclei of Goll and Burdach is only a sketch in its beginning. Of the cranial afferent dilatation paths only part of the trigeminus is drawn. The path of the acusticus, etc., is omitted in order to simplify the scheme.

ILLUSTRATING DR. LUTZ'S ARTICLE ON "THE LIGHT PUPILLARY REFLEX, ITS PATH, AND ITS ABOLITION CALLED IMMOBILITY OF THE PUPIL TO THE LIGHT REFLEX, AND REPORT OF A CASE OF UNILATERAL ARGYLL-ROBERTSON PUPIL, IN WHICH CONSENSUAL REACTION EXISTED IN BOTH EYES."







## III.

## THE TERMINAL ORGAN, THE IRIS.

After what we have demonstrated it is easy to understand that the terminal organ is under the constant influence of two antagonistic muscles: the sphincter and the dilatator muscles of the iris. During the light reflex we can observe not only the rapid, clonic, and isolated contraction of each of these muscles, but we can also see that the two antagonists are, as all muscles innervated by a cerebrospinal center, under the constant influence of a tonus, which is simultaneous for both eyes. Bach showed that pupillary dilatation through pain continues after section of the trigeminus, sympathicus, and ganglion cervicale superius, and disappears only after section of the oculomotorius. The influence of the two antagonists is even seen after death. In agony, the pupil is large, then it becomes small through the rigor mortis of the sphincter, which is stronger than the dilatator; later on, the pupil becomes large through action of the dilatator which survives longer, and at last, after death of all these muscles, there takes place another slight contraction of the pupil.

But not only are the nervous impulses responsible for this tonus; we have also to consider the elasticity of the tissue and the influence of the vasomotor nerves on the blood vessels of the iris known since the experimental injection of the blood vessels by Primelles (1840): diminution of the tension in the vessels, with less blood in them, produces dilatation of the pupil; increase of their tension, contraction of the pupil, through the radial structure of the iris. In this direction, the ganglion ciliare is of the utmost importance. Lapersonne tells us that we have to consider it at the same time as a cerebrospinal and as a sympathetic ganglion. It has no influence on the exterior muscles of the eye, and cannot be made responsible for irido-dilatation. It can only have influence on the ciliary muscle and on the sphincter. We know that burning of the cornea and iridectomy is followed by degeneration in the large ganglion cells of the ciliary ganglion, and we know from the experiments of Langendorf that even a certain time after death when excitation of the nervous oculomotorius is without effect



on the exterior muscles of the eye electrical excitation of the sensitive ciliary nerves produces irido-contraction. We can therefore divide the pupillary reflexes according to Vennemann into three classes:

1st. Peripheric reflex or vasomotor arc (sensitive nerves of the eye, large ganglion cells of the ciliary ganglion, ganglion cells of the ganglion Gasserii, short ciliary nerves).

(a) Hyperfunction shows miosis.

*e.g.*, in Angina phlegmonosa Vincentii, if soft palate is affected (nervus palatinus; nervus maxillaris superior; ganglion Meckeli; ganglion ciliare; nervuli ciliaris breves).

(b) Hypofunction shows mydriasis.

*e.g.*, through glaucomatous compression of the nervi ciliaris.

2d. Basal reflex, or light reflex arc.

(a) Hyperfunction shows miosis.

*e.g.*, through strong illumination.

(b) Hypofunction shows mydriasis.

*e.g.* darkness, amblyopia.

3d. Cortical reflex arc.

(a) Accommodation.

(aa) Hyperfunction shows miosis, *e.g.* hyperopia.

(bb) Hypofunction shows mydriasis, *e.g.* myopia.

(b) Psychical function, attention, etc.

(aa) Hyperfunction shows mydriasis.

*e. g.*, fright, strong attention, mania.

(bb) Hypofunction shows miosis.

*e. g.* newly born child, old people, idiocy.

The first one of the three reflexes is unilateral; the two others are always bilateral, consensual, and direct. The first two are always inconscient and involuntary; the last one is not conscient but can be influenced voluntarily.

#### IV.

##### PHARMACOLOGY OF THE PATHS OF THE LIGHT REFLEX.

We divide the nerves, from the pharmacological standpoint, into two groups:

1. Animal nerves: they leave the brain or the cord in an

uninterrupted series and proceed directly to the terminal organ. They innervate the voluntary organs.

2. Vegetative nerves: they leave brain and cord only from circumscribed areas; they never go directly to the terminal organs but always enter in their path in connection with some peripheric ganglion. They can pass through different ganglia, but find the real nervous connection in only one of them; they go to the organs that work automatically. They are subdivided into two groups:

*a.* Parasympathetic nerves: they go direct from the brain to the peripheric ganglia. The parasympathetic nerve of the mesencephalon is the nervus oculomotorius.

*b.* Sympathetic nerves: they leave the cord through the white rami communicantes, enter the truncus sympathetitus, leave it as gray rami communicantes, and join the spinal nerves.

This division is important for the susceptibility of certain poisons on the nerves, *e. g.* nicotine is a poison for all vegetative nerves, but it never attacks them in their path, but only in the place where they enter into real nervous connection with the last neuron. It is therefore the best means of determining the terminal ganglia of all the vegetative nerves, the parasympathetic as well as the sympathetic nerves. On the other hand, adrenaline excites only the terminal fibers of the sympathetic; it never acts on parasympathetic nerves. The same action which adrenaline has on the sympathetic termination takes place with physostigmine and muscarine on the parasympathetic nerve terminals. The parasympathetic terminals which physostigmine and muscarine excite, are paralyzed by atropine or curare. Meanwhile, muscarine excites the parasympathetic nerves peripherically, picrotoxine and camphor excite parasympathetic nerves only in their central part.

Considering these pharmacological effects and remembering the above-named physiological experiments, we find:

#### *A. Dilatation of the Pupil.*

1. Through central paralysis of the sphincter centrum, *e. g.* in asphyxia or botulismus.

2. Through central irritation of the dilatator center, *e. g.* in fear, pain, concentration of thought, etc.

3. Through local application of nicotine to the ganglion ciliare.
4. Through peripheric paralysis of the parasympathetic nervous terminals by atropine.
5. Through peripheric excitement of the sympathetic nervous terminals by adrenaline.

*B. Contraction of the Pupil.*

1. Through central paralysis of the inhibitions which dilate the pupil, *e. g.* in slight narcosis which eliminates all sensitive and sensorial irritations or in morphine narcosis.
2. Through central irritation of the sphincter centrum by picrotoxine.
3. Through local application of nicotine to the ganglion cervicale superius.
4. Through peripheric excitement of the parasympathetic nerves by eserine.

The afferent sensory pupillo-motoric path could only be influenced by strychnine, which facilitates the transmission of the stimulus from one neuron to the other.

V.

THE ABOLITION OF THE LIGHT REFLEX (CALLED "ARGYLL-ROBERTSON PHENOMENON").

The movements of the pupil can be altered in many different ways, but the most interesting changes are condensed under the name of ARGYLL-ROBERTSON'S PHENOMENON. This abnormal reaction of the pupil is called after its first describer, who published (1869) some cases of diseases of the spinal cord. Basing his conclusions on five patients, he describes the phenomenon as consisting of the following different symptoms: the pupil does not react on light incidence; it reacts very well on convergence and the pupil shows a marked miosis. These observations were confirmed soon afterwards by H. Knapp. Argyll-Robertson was right in so far as further investigation showed the utmost importance of this pupillary reaction for the whole pathology, which is perhaps best illustrated by the

investigations of Mann, who found the Argyll-Robertson phenomenon in tabes even more constant than the Westphal phenomenon. But he was not right in describing the miosis as an integral part. Uhthoff (1914) tells us that miosis is found in only about 30% of the cases; that the others may lack miosis, or can even show mydriasis. Erb had already expressed, long before, the opinion that miosis and immobility of the pupil to light are two different symptoms caused by two different processes. Miosis is found more in tabes, mydriasis more in paralysis generalis. Wilbrand-Saenger called attention later on to a certain atrophy of the iris, which is found from time to time together with immobility to light, and which Dupuy-Dutemps (1905) believes so characteristic that according to him it alone permits the diagnosis of the Argyll-Robertson phenomenon. Finally, Bach stated that together with immobility to light, there is mostly found an abolition of the pupillo-dilatation reflex through excitation of the trigeminus. The phenomenon is called to-day by most authors "immobility of the pupil to the light reflex," and its modern definition is given by Axenfeld in the following terms: "We speak of pure immobility of the pupil to the light reflex, when we find in eyes whose vision is intact, the pupils immovable to light incidence in the same as well as in the fellow eye; during convergence the pupils become miotic, and that in a very marked manner; these eyes lack almost regularly the reflex dilatation through pain or psychic irritation." Winaver Bronislas expresses himself in the same way, but adds a restriction: "Under the condition that there exists no lesion in the eyeball or in the optic nerve."

The importance of the immobility of the pupil to the light reflex lies in its connection with certain diseases which are the consequence of syphilitic infection. It is the special merit of Babinski to have pointed this out. It has never been found in healthy persons, *e. g.* Charpentier and Etchevery investigated 1100 soldiers without finding a single case. We also find absence of the light reflex generally with mydriasis in certain abnormal stages, such as in epileptic attacks, hysterical crises, syncope, catatonic stupor, extreme fright, and excessive muscular consumption; but it is not certain that all these cases are genuine cases of immobility of the pupil to the light reflex. They can be explained as cases of spastic mydriasis. Doubt-

less, we find the typical Argyll-Robertson's phenomenon very exceptionally in certain non-syphilitic diseases, which are: sclerosis multiplex; syringomyelia; atrophía muscularis spinalis progressiva; neuritis interstitialis hypertrophica; ataxia hereditaria; alcoholismus chronicus gravis; and diabetes; oftener, in certain traumatisms of the skull. But these are all rare exceptions, and Grasset (1912) is right when he expresses the importance of the immobility of the pupil to the light reflex for the practitioner in the following terms: "The Argyll-Robertson phenomenon indicates tabes, sometimes general paralysis, always previous infection with syphilis." If we exclude the above-named exceptions where syphilis could not be detected even through the most modern methods of investigations of the blood and cerebrospinal fluid, and even in autopsies, we find immobility of the pupil to the light reflex as a consequence of syphilitic infection in first line in tabes: in incipient tabes it is found in 45% of the cases; in the fully developed, in 85%; in second line, we find it in paralysis generalis—in 17% of the cases in adolescent, and in 50% of them in adults; finally, we find it in about 10% of cases of cerebral syphilis (*e. g.* syndrome of Babinski). The immobility of the pupil to the light reflex can precede a metasymphilitic disease for several years, and can be during that time the only sign of syphilitic infection. It develops always very slowly and after having appeared it, as a rule, remains permanently. But there are also known some rare cases (*e. g.* Eichhorst) which show intermittence, which means that the immobility of the pupil to the light reflex disappeared and reappeared, even several times, under treatment, or without it. It can also develop as a sequel of a paralysis of the nervus oculomotorius in persons where no other sign of tabes or paralysis generalis could be observed. Intermittence is found in all forms, but is mostly observed in cases of cerebral syphilis. The Argyll-Robertson pupil is found with preference in the later stages of syphilis; but it can also appear exceptionally in the first period of the infection: Sulzer (1901), *e. g.*, observed a case about one year after the appearance of the chancre.

The immobility of the pupil to the light reflex is found almost always in both eyes, even when there exists a marked anisocoria, miosis on one side and mydriasis on the other. In rare cases

the phenomenon is found on only one side, and these unilateral cases of Argyll-Robertson's pupil are mostly precursors of general paralysis. All authors agree that a unilateral immobility of the pupil to the light reflex has absolutely the same prognostic significance as its bilateral form—which means that a man with a unilateral Argyll-Robertson is never a healthy man, and is probably a candidate for a severe affection of his nervous system. It is therefore of the utmost importance to make in every case of anisocoria the examination of the light reflex. The unilateral form is very rare. I have tried to collect all the publications on unilateral cases found in literature, and have added them to the end of this article. Its rareness is perhaps best illustrated by the fact that in 1900, in a scientific session in Paris, where Babinski presented a case of unilateral Argyll-Robertson, men such as Déjerine and Gilbert Ballet declared having each seen only one case in their lives. Bach saw one unilateral case during several years as the only symptom of an incipient tabes. Sauvinau saw one case disappear under treatment with mercury. Rothmann, Abelsdorf, and Frenkel-Garipuy saw one unilateral case as a sequel to an improved case of oculomotor palsy. Tanzi saw in a 50-year-old patient with beginning dementia paralytica a unilateral Argyll-Robertson complicated with miosis appear and completely disappear five times during the same year. Unilateral Argyll-Robertson is mostly found in dementia paralytica where we see it in about 3 to 5% of all cases with immobility of the pupil to the light reflex. In the other diseases it is observed less often, and it is natural that it is found relatively oftener in traumatic cases, *e. g.* Krüger, Axenfeld, Weste. In trauma with compression on the brain it can also appear as a transitory phenomenon with loss of consciousness, and it is then to be considered as a very bad prognostic sign. It is also worth while mentioning the experience of Dufour who saw a woman with a slight Westphal, anisocoria, and unilateral Argyll-Robertson, and no other symptom from the nervous system; the patient died accidentally soon afterward, and the autopsy showed all the typical lesions of the whole spinal cord which we are accustomed to find in tabes incipiens.

Grasset gives the following definition of unilateral immobility of the pupil to the light reflex: "The affected pupil re-

mains immovable to light incidence in the same as well as in the fellow eye; the healthy pupil reacts to light incidence in the healthy eye as well as in the affected one." This means that in the affected eye the direct light reflex and the consensual one are abolished. It represents the common form of unilateral Argyll-Robertson. But there are some other observations, as those of Babinski, Bechherew, Berger, Jessop, and Piltz, which show the same peculiarity as my own observation which gave occasion to this study, namely, that the direct light reflex is abolished alone and that the consensual light reflex is found present in both eyes. Such cases represent therefore a pure direct light immobility and the common form of Argyll-Robertson represents a total light immobility (direct plus consensual). One could conclude from this distinction between pure and total light immobility, that we could also find the bilateral form of pure light immobility, that is to say, cases where on both sides the direct light reflex is abolished, but not the consensual one. I do not know if there exist any publications bearing out this point. It seems to me impossible with regard to the above-named description of the last step of the afferent pupillo-motor path. But we must remember how easily we forget to detect the presence of the consensual reaction in bilateral forms of Argyll-Robertson. On the other hand, the above-mentioned sketches give us the explanation that it is theoretically possible to find cases where in both eyes the direct light reflex is present and in both eyes the consensual reaction is abolished. This shows once more how important it is to always make the examination on consensual reaction.

*Personal Observation.* Mr. J. P., 51 years old, merchant, Spaniard, was sent to my office by his family physician, Dr. C. Desvernine, for an anisocoria which was observed accidentally. His father had died from Bright's disease; he had never had syphilis; his mother died of diabetes; one brother died of diabetes before he was 30 years old; two sisters suffer from polyuria, and one sister from hepatic colic. His wife died from diabetes; she had one abortion, no children. The patient has never been ill and denies absolutely any syphilitic infection; Wassermann reaction of the blood is negative. He consulted his physician for a slight paresthesia in his right hand. He had always shown a peculiar character, and in later times has held some very queer

ideas, with a certain mixture of delirium as to wealth. Upon his wife's death he wished to erect a mausoleum in which a modern bathroom and toilet should be installed, as well as an expensive harmonium.

STATUS: 8—IX—1916. Paresthesia in the right hand, diminution of the patellar reflex on both sides. Vision: R. & L. Eye  $\frac{4.5}{18.0}$  plus 2.25 =  $\frac{4.5}{4.5}$ . Media quite transparent, fundi normal. No diplopia. Marked anisocoria. No ptosis, no enophthalmus, no alteration of the vasomotor innervation. Right pupil: 1.5mm, Left pupil: 4.5mm. Left pupil: reacts directly on light incidence; consensually on light incidence; on convergence; on accommodation; and on excitation of the trigeminus. Right pupil: absolutely no reaction on direct light incidence; doubtful (consensual) on incidence in the other eye; very well on accommodation; very well on convergence; absolutely no reaction on excitation of the trigeminus.

14—X—1916. Dark room: R. P. : L. P. : 1.5 : 3.5mm. Ordinary illumination: R. P. : L. P. : 2.0 : 4.5. Reaction on trigeminus abolished on the left eye.

20—XII—1916. Patient had some mercury injection. Dark room: R. P. : L. P. : 2.0 : 3.0. Ordinary illumination: R. P. : L. P. : 2.5 : 4.0. Anisocoria diminished.

Trigeminus reaction reappeared in the left eye, absolutely clear. In the right eye it cannot yet be detected with certainty. The right pupil reacts slightly consensually on incidence in the left eye, and after staying for a while in the dark room, one can also see a very slight direct light reaction in the right eye.

15—III—1917. Dark room: R. P. : L. P. : 2.5 : 3.5. Ordinary illumination: R. P. : L. P. : 3.0 : 4.5. Left pupil reacts: On direct light incidence; consensually, on light incidence, on the other eye; on convergence; on accommodation. The reaction on trigeminus cannot exactly be detected. R. P. reacts: Doubtfully on direct light incidence; markedly consensually on incidence in the other eye; very well on convergence and accommodation. Trigeminus reflex cannot be detected with certainty.

19—IV—1917. Status: *idem*.

24—X—1917. Status: absolutely the same. Consequently, the patient showed during one year the abolition of the direct light reflex in one eye, whilst the consensual light reaction could be detected in both eyes.

The unilateral cases of Argyll-Robertson have their special and utmost importance in the study of the anatomical founda-



tion of the immobility of the pupil to the light reflex. To explain this, let us consider the different theories and opinions which tend to fix precisely the situation of the anatomical lesion. Different authors have located the lesions in:

(a) *The spinal cord* (efferent pupillo-dilatation path). It was originally the idea of Argyll-Robertson that the cause of this phenomenon was located in the spinal cord. He based his opinion in the miosis and in the fact that the pupils dilate so slowly on applications of mydriatics. This theory, especially defended by Rieger, Forster, and Reichardt, is even sustained to-day (1912) by Siebert and Harald. They base their opinion on the belief that it cannot be solely by chance that we almost regularly find degenerations there. In favor of their opinion we find especially the case of Remak, where there existed besides, an atrophía muscularis spinalis progressiva.

Against this theory we have to remember that miosis is not typical for immobility of the pupil to the light reflex and that interruption of the neuron which precedes the centrum ciliospinale, and which is followed by miosis, can never explain the abolition of the light reflex. Furthermore, we must remember that Déjerine could not find any alteration in the spinal cord in cases with very marked bilateral Argyll-Robertson.

(b) *Ganglion ciliare* (efferent pupillo-contraction path). This idea of Van Gehuchten has been especially defended by Marina and Lafon. Marina bases his opinion on bibliographic and anatomical studies which convinced him that there were many cases of Argyll-Robertson in which no changes were found in the region of the pupillo-motor centers, and further, on his anatomical investigations in the ganglion ciliare which showed him that in any case of immobility of the pupil to the light reflex, degenerations could be found in the ganglion cells of the ganglion ciliare, and that these degenerations were absent in people with normal pupil reaction. Especially one case of unilateral Argyll-Robertson, in which degenerations were only found on the unhealthy side and not in the healthy one, convinced him in this respect. Immobility of the pupil to the light reflex would therefore, according to this theory, be a flaccid paralysis of the efferent pupillo-contraction path, and one could also be tempted to cite in favor of this theory the iris atrophy described by Dupuy-Dutemps.

Against this opinion we must bear in mind that paralysis of the ganglion ciliare with nicotine is followed by pupillo-dilatation and complete paralysis of the pupil. The action of nicotine shows very clearly that we cannot consider the fibers for accommodation as animal fibers of the nervus oculomotorius crossing the ganglion ciliare without entering there into communication with the ganglion cells, and consider on the other hand the light fibers as vegetative fibers of the nervus oculomotorius which do not go directly to the iris, but enter in their route into communication with the short ganglion cells of the ciliary ganglion. Nicotine conduces to paralysis of all efferent fibers of the ciliary ganglion, and it therefore does not explain why the fibers of the light reaction do not work, while the fibers of convergence and accommodation act very efficiently in cases of Argyll-Robertson pupil.

Lesions of the short ganglion cells of the ciliary ganglion could not explain either why we find miosis in about 30% of the cases; in others we find normal pupils or even mydriasis. Paralysis of the short ganglion cells must be followed by dilatation of the pupil.

We have further seen that in some unilateral cases the consensual reaction can be found in both eyes, and I think it more natural to accept that the same efferent fibers conduct the excitation of the direct as well as of the consensual light reflex.

We cannot either consider the iris atrophy of Wilbrand-Saenger as favorable to a flaccid palsy—because Dupuy-Dutemps observed that the atrophy can precede the apparition of the immobility to light.

A lesion in the ciliary ganglion would not explain, either, the abolition of the dilatation reflex following excitation of the trigeminus, which we find so often together with immobility to light, because the efferent pupillo-dilatation path does not enter in nervous connection with the ganglion cells of the ciliary ganglion as shown by the nicotine experiments of Langley and Anderson.

As a rule, we find the Argyll-Robertson bilateral; only exceptionally is it unilateral. This argues likewise against the opinion of Marina. For, if he were right, unilateral Argyll-Robertson should be found oftener than the bilateral, because

we must admit that there is more probability of only one ciliary ganglion being affected.

Against the opinion of Marina we have also to mention the investigations of Andre Thomas (1910) who made autopsies in three cases of Argyll-Robertson; he could not find any degeneration in the short ciliary nerves nor in the ciliary ganglion, nor in the roots which the ciliary ganglion receives from the oculomotorius and the trigeminus.

The fact that we find the immobility of the pupil to the light reflex as a rule bilaterally and only exceptionally unilaterally is also a reason why we cannot expect to find the anatomical substratum of the Argyll-Robertson in the nervus oculomotorius (efferent route) or in the retinae, nervi optici, corpora quadrigemina anteriora (sensory afferent route). It forces us to search for the lesion in the immediate neighborhood of the centers.

(c.) *Centers:*

1. Fascicular lesions. It was Heddæus who first thought that Argyll-Robertson was produced by a fascicular lesion of the efferent fibers of the photomotor center. He based his opinion on the cases with mydriatic light immobility of the pupil.

Against this opinion we find miosis in about 30% of the cases. Interruption of the efferent fibers is followed by pupillo-dilatation.

Against his opinion we find further the few cases in which the consensual reaction was found in both eyes. As there does not exist a decussation of the efferent fibers, a fascicular lesion nearly always abolishes the direct and the consensual light reflexes at the same time.

2. Nuclear lesions. The opinion that Argyll-Robertson is produced by a nuclear lesion has been defended in first line by Levinsohn.

The fact that precisely characterizes the immobility of the pupil to the light reflex, namely, that the pupil can be contracted voluntarily very well in convergence and accommodation, is an argument against this opinion. We know further that nuclear lesion is followed as a rule by mydriasis.

3. Supra-nuclear lesions. Since pupillo-dilatation is only lacking in light reflex but not in convergence or accommodation

reflex, we have to look for the interruption where the light excitation has not yet reached the centers for pupillo-contraction and pupillo-dilatation. This speaks in favor of a lesion in the communication between the corpus quadrigeminum anterius and the photomotor centers. This opinion has been especially defended by Landois, Déjerine, v. Monakow, and Uhthoff. Our considerations as to the path of the light reflex showed us that an interruption of the communication between the corpus quadrigeminum and the nucleus photomotoricus must be followed by abolition of the direct and consensual light reflexes, and will remain unilateral if the lesion remains unilateral; but as these neurons run each very near the other in the neighborhood of the third ventricle, we can also understand that even small lesions must abolish the light reflex in both eyes. In favor of this opinion may be cited: the cases of syringomyelia complicated with unilateral Argyll-Robertson; the traumatic case of Pope who found at autopsy the third ventricle full of blood; the traumatic cases of Guillain, Rochon-Duvigneaud, and Trosier. Furthermore, anatomical investigations of many authors who found in the gray matter itself, around the third ventricle, degenerations of the fibers, blood extravasations, and obstruction of the vessels. In favor of this localization we have, furthermore, the case of Moeli (*Archiv f. Psychiatrie*, xviii.), who found the lesion of a bilateral Argyll (with intact vision, intact mobility of the eyeballs, and good convergence reflex) to be a tumor involving both sides of the posterior walls of the third ventricle. Von Monakow (*Archiv f. Psychiatrie*) found in a case of one unilateral loss of the pupillary light reflex a small sclerosed focus in the tissue between sphincter nucleus and the external geniculate body. Such a localization of the anatomical lesion would also agree closely with the fact that tabes is preferably a disease of the afferent sensitive routes. Paralysis and tabes are clearly characterized by the early degenerations of the last termination of the sensitive nerves.

This theory would also better explain how the communication of the sensory-afferent way and the efferent pupillo-dilatation way is interrupted in both eyes. For we must remember that in cases of Argyll-Robertson there is not only loss of the influence of light in the pupil, but also of the influ-

ence of the sensory excitations through trigeminus and the psychical excitations through the cortex. From anatomical researches and clinical observations in cases of hemiplegia Déjerine deduced that the sympathetic path descends to the mesencephalon. We can therefore say that the lesion which cuts the communication between sensory afferent path and pupillo-contraction center cuts at the same time the communication between the sensory afferent path and the sympathicus of the mesencephalon.

One could be tempted to say that the miosis argues against a lesion in this last afferent sensory neuron. This argument is eliminated by the fact that amaurosis of both eyes, *e. g.*, through atrophía optica tabetica is without any influence on the form of the light immobile pupil, that is, the miotic pupil remains miotic.

Lafon (1909) called attention to the importance of studying the modifications of the Argyll-Robertson pupil consequent upon lesions of the different parts of the afferent and efferent paths of the light reflex way. These are:

I. ARCUS SENSORIO-MOTORICUS.

(a) Lesion of the afferent sensory path, *e. g.*, amaurosis is without any effect on the pupil.

(b) Lesion of the efferent pupillo-contraction path:

1. The action of atropine is retarded.
2. Compression of the post-ganglionic fibers, *e. g.*, by glaucoma, produces dilatation of the pupil.
3. Paralysis of the oculomotorius also produces a dilatation of the pupil, but not so strong as in uncomplicated cases.

II. ARCUS SENSITIVO-MOTORICUS.

We do not yet know the influence of destruction of the trigeminus or of the ganglion cervicalis superius.

III. ARCUS CORTICALIS.

(a) It is true that abnormal excitations of the cortex as we see them, *e. g.*, in maniacal people, are without influence on the Argyll-Robertson pupil.

(b) We do not yet know the influence of the hypofunction of the cortex on the Argyll-Robertson, *e. g.*, in sleep, in syncope, or narcosis, that is, if the pupil grows smaller.

From this observation we can conclude that the lesion of the afferent pupillo-contraction path is without influence; that the

lesion of the cortical inhibition path is probably also without influence; that we do not know anything certain about the influence of the afferent and efferent pupillo-dilatation paths; and that finally lesions on the efferent pupillo-contraction path have doubtless an influence on the Argyll-Robertson pupil. We need further observations in this direction.

Erb holds that light immobility and miosis are two different symptoms produced by two different processes. But it seems to me more natural to accept that the appearance of miosis depends more on the extension of the lesion. We can accept that miosis is produced by the lesion of the mesencephalic sympathicus or by the interruption of the last sensory afferent neuron with this mesencephalic sympathetic. This miosis differs fundamentally from Horner's syndrome in that it lacks ptosis, enophthalmus, and alterations of the vasomotor innervation, but we can consider that this difference is caused by the fact that in Argyll-Robertson pupil the neuron is interrupted before the centrum cilio-spinale, and that in Horner's the same path is interrupted in the neuron whose origin is found in the ganglion cells of the centrum cilio-spinale. Levinsohn (1908) has especially studied the phenomenon of miosis; he called attention to the fact that in Horner's syndrome, miosis disappears in a dark room, notwithstanding the existence of a paralysis of the sympathetic, and that in Argyll-Robertson pupil, darkness is without any influence, which is easily understood by the complication of light immobility and which best shows that miosis and light immobility are two different things. Furthermore, miosis is found very much more pronounced in the Argyll-Robertson pupil than in Horner's syndrome. We cannot explain the miosis by a lesion of the afferent sensitivo-motor path, because we find many cases with marked miosis where the sensibility is entirely normal, and on the other hand many cases without miosis where the sensibility is abolished. The unilateral cases of Argyll-Robertson show that we cannot localize the cause in the afferent sensory route (retina; nervus opticus; corpus quadrigeminum), because such lesions should conduce to bilateral miosis. As, furthermore, the fibers of pupillo-contraction for light incidence and for accommodation as well as convergence are intimately intermixed in the nervus oculo-

motorius, we cannot expect to discover the cause of the miosis in the efferent pupillo-contraction path. These arguments induced Levinsohn to search for the lesion in the nucleus, and he admits that in the nucleus sphincter there are two parts: one which is responsible for the pupillo-contraction on light incidence, and another for pupillo-contraction through accommodation, convergence, etc.; if the first part is destroyed, we have immobility of the pupil to the light reflex; and through irritation of the second part miosis; if the destruction extends to the other part, we have total immobility of the pupil with mydriasis. Against this theory we should remember that miosis can persist for several years and it would be very improbable that during this whole time we would have miosis through pure irritation of the area; further, if the miosis were produced by irritation, it should always be complicated with spasm of the accommodation (*musculus ciliaris*). This latter has never been observed, and we see therefore, that the study of the miosis also forces us to search for the lesion not in the centers but in their neighborhood, that is, in that region where the afferent path joins with the efferent pupillo-dilatation path. An interruption of the connection of the afferent sensitive pupillo-dilatation path with the photomotor center explains only the loss of the trigeminus dilatation reflex. I think that the marked miosis is produced by the interruption of the *fibra descendens fascic. longit. post.*, which goes from the *corp. quadrig. ant.* to the *centrum cilio-spinale*. If the lesion destroys the crossing of Meynert (Meynert's *fontänen-artige Haubenkreuzung*) just in the median line, it produces bilateral miosis, if it acts only on one side, unilateral miosis follows. If the lesion is to be found there, it would explain at the same time why we find so often light immobility and miosis combined. A lesion of the *fibra descendens fascic. longit. post.* explains also the presence of miosis in diseases of the spinal cord, without that light immobility is found at the same time. This leads me to believe that the light immobility is produced by interruption of the fibers (of the *fascic. tectobulbaris*) which go from the *corpus quadrig.* to the *nucleus photomotoricus* and the miosis by the interruption of the fibers (of the *fasciculus tecto-spinalis*) which go from the *corp. quadrig. ant.* to the *centrum cilio-spinale*.

With regard to the foregoing considerations on the route of the light reflex and with regard to the various clinical observations and pathological discoveries in cases of immobility of the pupil to the light reflex, I think that we cannot accept the argument of some authors, that the Argyll-Robertson phenomenon can be produced in different ways by anatomical lesions in different parts of the light reflex path. I think that the lesion must always be found in the stage between the corp. quadrig. and the nucleus principalis lateralis.

#### SUMMARY.

##### A. Description of the light reflex:

1. The light reflex consists in a contraction of both pupils on increase of illumination, and in bilateral pupillary dilatation on reduction of illumination. Only light incidence on the retina reduces contraction of the pupil, while irritation of all other cranial and spinal nerves is followed by irido-dilatation.

2. The adaptation of the retina is of great importance for the amplitude of the pupillary reaction. Perhaps the amacrine cells of the centrifugal neuron chain are responsible for this.

3. The size of the pupil is in inverse proportion to the square root of the light intensity (Ovio).

4. The light reflex begins at the end of the fifth foetal month and is not abolished, in healthy persons, through age.

##### B. Path of the light reflex:

1. The light strikes the cones and the rods of the whole retina. The pupillo-motor effect of it is stronger in the macular region, and decreases rapidly towards the periphery. The multi-stratified bipolar cells of the second retinal neuron conduct the light irritation from different cones and rods to the multi-stratified ganglion cells of the third retinal neuron. The fibers of these latter cells conduct the light stimulus in the thick fibers through opticus, chiasm, where they perhaps do not undergo the semi-decussation of the visual fibers, and through the tractus opticus. Hemikinesis cannot be regarded as proof of the semi-decussation of the pupillo-motoric fibers in the chiasm. In front of the corpus geniculatum laterale the pupillary fibers enter the brachium conjunctivum and go through the same to the ganglion cells in the deeper layers of the



corpus quadrigeminum anterius. The fibers of these last cells reach, through fasciculus tecto-bulbaris, the nucleus photomotoricus; part of them keep on the same side and produce the direct light reaction; collaterals of them go on the other side and produce the consensual light reaction. These central semi-decussations take place in the neighborhood of the nucleus photomotoricus.

2. As *center* can be regarded the frontal pole of the nucleus lateralis principalis (von Monakow). But, it seems to me more natural to regard as pupillo-motoric center the deeper layers of the corpus quadrig. anterius, from which originate the two efferent pupillo-motor paths: firstly, for active pupillo-contraction, through nucleus principalis lateralis, and secondly, for active pupillo-dilatation through the centrum cilio-spinale.

3. The *efferent pupillo-contraction path* begins in the nucleus oculomotorius. The fibers of the photomotor center enter without crossing the nervus oculomotorius, and reach, through the short root, the small ganglion cells of the ciliary ganglion. The fibers of these latter reach, through the short ciliary nerves, the musculus sphincter iridis.

4. The *pupillo-dilatation* is produced by irritation of all cranial or spinal nerves except opticus, and by central inhibition. Its irritation produces inhibition of the photomotor center (responsible for pupillo-contraction) through the ascending fibers of the fasciculus longitudinalis posterior, and produces on the other side active pupillo-dilatation through the descending fibers of the fasciculus longitudinalis posterior. These latter fibers reach the centrum cilio-spinalis in the spinal cord. Its fibers go without crossing through the ventral roots of the last cervical and the first two thoracic nerves as rami-comunicantes to the truncus sympathicus cervicalis and to the ganglion cervicale supremum. The fibers of this ganglion reach, through the anterior branch of the Gasserian strand, the ophthalmic nerve and the long ciliary nerves, the musculus dilatator iridis. These latter fibers have no connection with the ciliary ganglion.

5. The iris stands under a constant tonus of the nervus oculomotorius as well as of the nervus sympathicus (long ciliary nerves). It is furthermore constantly influenced by the

large ganglion cells of the ciliary ganglion, which received stimuli from the Gasserian ganglion (*radix longa*) and the plexus sympathicus carotideus (*radix sympathica*) or vasomotor branch of the *truncus sympathicus cervicalis*.

6. The light reflex—the direct as well as the consensual one—is a basal reflex, and does not go through the ciliary ganglion.

C. Immobility of the pupil to the light reflex:

1. The Argyll-Robertson phenomenon consists in the abolition of the direct and consensual pupillary reaction to light incidence in both eyes, while the reaction on convergence is found normal. It is mostly connected with abolition of the pupillo-dilatation reflex on irritation of the trigeminus. In 30% of the cases we find also miosis.

2. It is one of the most important indications of diseases following syphilitic infection, and it can also be found exceptionally in other diseases. In rare cases the Argyll-Robertson phenomenon is found only on one side. The affected eye then shows no light reaction: its pupil does not contract on incidence of light in the same as well as in the fellow eye.

3. To explain the phenomenon we have not to look for the lesion in the spinal cord, nor in the ciliary ganglion, but in the region of the photomotor center: the interruption of the connection between the last afferent sensory step and the photomotor center explains the immobility of the pupil to light incidence; if the lesion is large enough it interrupts the connection between *fasciculus tecto-bulbaris* and *nucleus principalis lateralis* on both sides; otherwise it cuts it only on one side. Minor lesions only interrupt the connection of the direct light reaction path and leave untouched the collaterals going to the other side; in such cases the consensual reaction is found present in both eyes.

Miosis can be explained by the interruption of the first neuron for active pupillo-dilatation (*fibra descendens fasciculi tecto-spinalis*).

The loss of the trigeminus reflex can be explained by the interruption of the connection between *fibra ascendens fasciculis longitudinalis post.* (trigeminal part) and the photomotor center; the lesion eliminates the inhibitive influence of the afferent pupillo-dilatation path.

I owe part of the literature to the kindness of Dr. M. Bango and Dr. C. E. Finlay, and most especially to Dr. José Valdés Anciano, Professor of Neurology at the University of Havana. I take pleasure in expressing to them my thanks for affording me access to their libraries.

### CORRIGENDA

I have to correct the opinion (expressed in the first part of this paper) that the crossing of the pupillary fibers in the chiasm has not yet been proven. The semi-decussation of the pupillary fibers in the chiasm has been demonstrated anatomically in cats by S. Ramón y Cajal, and in man by Van Gehuchten. Histological researches of the brachium conjunctivum ad corp. quad. ant. showed that this consists of two different bundles: 1. One which comes down from the lenticular region and probably therefore from the cortex, the fasciculus cortico-bigeminalis; it ends in descending lateral dendrites in the deepest layer of the corp. quad. ant. 2. The other comes from the optic tract and ends in ascending dendrites in the next layer higher up. Ramón y Cajal could demonstrate that the latter receives the majority of the fibers from the opposite eye; Van Gehuchten believes that two thirds of all pupillary fibers cross the chiasm. These anatomical findings fully explain the experiments of Trendelenburg in cats and especially the very exact observation in man by Pierre Marie and Chatelin.

If we ask ourselves the reason why hemikinesis can be observed far more easily in animal experiment, we must remember that the lower animals have no visual cortex. Corp. geniculatum externum, pulvinar and calcarine fissure become important only in mammalia. On the other hand, the corp. quad. ant. have in the lower animals a far more complicated structure, and are so large that anatomists speak of a tectum opticum. This leads us to the conclusion that the pupillo-motor function of the corpus quad. ant. in man is not a function specifically different from that of the visual cortex,—but only the most primitive or phylogenetic oldest form of the visual function. Therefore, we can no more hold the opinion that the fibers of the radiation of Gratiolet have no pupillo-

motor effect; on the contrary physiological experiments have shown that electrical excitation of the calcarine fissure produces a marked miosis. As a consequence we shall find hemikinesis not only in lesions of the frontal visual neuron (optic tract) but also in those of the occipital neurons. This explains to us the observations of hemikinesis in hemianopsia posterior made by Clifford B. Walker and Déjerine. Furthermore considering the fact that in man the corp. quad. ant. are only very small compared with other visual centers, we can understand why hemikinesis through lesions of one optic tract is far more difficult to observe in man than in animals, because the clinical appearance of the hemiopic pupillary reaction is hidden by the action of the pupillo-motor fibers which descend from the opposite hemisphere and partially cross with the fibers of the fasciculus tecto-bulbaris. This consideration may explain to us, furthermore, why have we not definite clinical observations of hemikinesis without hemianopsia, such as we can expect from a lesion of the brachium conjunctivum. Such an occurrence would be concealed through the action of the pupillo-motor fibers descending from both cortices.

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WILBRANDT-SAENGER: *Neurologie des Auges*.

## THE INFLUENCE OF FUNCTION UPON THE STRUCTURE OF THE EYE.

BY DR. EDUARD UHLENHUTH, NEW YORK.

(From the Laboratories of The Rockefeller Institute for Medical Research.  
Author's Abstract.)

**I**F an eye of a larva of the European fire salamander (*Salamandra maculosa*) is severed from the optic center and is grafted, with a flap of surrounding skin, to another larva of the same species, the retina of the grafted eye undergoes a more or less severe degeneration, but after a short time begins to regenerate and the normal condition of the retina may be re-established completely. In the most severe cases the entire retina is destroyed with the exception of the most peripheral parts. The nuclei are broken down and form large masses of detritus which fill the space of the destroyed retina. The most interesting phenomenon during this process of degeneration is the behavior of the cells of the retina pigment epithelium. The pigment epithelial cells detach themselves from the pigment epithelium and migrate freely into the detritus masses; here they assume a phagocytic function and it is due mainly to these cells that the masses of detritus are quickly removed from the eye. Their activity lasts, although in a considerably diminished degree, even during the process of regeneration.

Regeneration is brought about mainly by mitotic divisions in the area of the retina next to the destroyed part; mitotic divisions are found in the inner and outer granular layers as well as in the ganglionic layer. Only after very mild degener-

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<sup>1</sup> Abstract of paper read before Section of Ophthalmology, New York Academy of Medicine, Feb. 18, 1918. The complete paper will appear in the *Biological Bulletin*.

ation the loss of the few degenerated cells is compensated by mitotic divisions of the cells of the pars retinae ciliaris only. In the beginning the part regenerated in place of the destroyed area of the retina is an undifferentiated mass of cells. Later on an epithelial-like granular layer is found which by regeneration of the fibrous layers from the old part into the new part is differentiated into the inner and outer granular layers and into the ganglionic layer. Finally the rods and cones regenerate from the outermost cells of the outer granular layer. At this time the activity of the pigment cells is stopped entirely.

However completely the structures of the grafted eyes may be restored, these eyes are unfit to function since no stimuli can be transmitted to the brain; for the grafted eyes are severed from the visual centers. Since despite this lack of function the functional parts of the eye, viz.: the cell elements of the retina, the rods and cones, which in regard to the specific function of the eye appear to be the most highly specialized parts of the eye, and in many cases the nervous structures may remain normal, it is clear that the opinion of many anatomists and surgeons that the structures of an organ cannot regenerate without the organ being in active function is erroneous. The structure of the grafted eyes not only regenerates, but also persists without the aid of function. Eyes preserved three and a half years after having been grafted show all the functional elements of the retina present.

Moreover, the same result can be obtained, if the functional stimulus, viz.: any trace of any kind of light is prevented from reaching the grafted eyes. Two curves are shown which demonstrate that the retinae recovered from their state of degeneration without light and secondly regenerated in darkness just as rapidly as in light.

Since the grafted eyes are severed from the nerve centers, the experiments also prove that a trophic stimulus, exerted by the nerve centers upon the tissue, is not a factor involved in the regeneration and maintenance of the functional structures of the eye.

It can be shown by a number of facts that the degeneration of the grafted eye is due to the interruption of the blood circulation; the restoration of the normal conditions in several parts of the eye can be watched with a binocular and it is

observed that degeneration stops and regeneration begins as soon as the newly established blood circulation reaches the part in question. Degeneration and regeneration in the grafted eyes are matters of nutrition.

The phenomena exhibited by the cells of the pigment epithelium are such as are known to occur in retinitis pigmentosa. Many of the grafted eyes show a very considerable retinitis pigmentosa, caused by the migration of the pigment cells into the retina and there many of these cells break down and deposit their pigment. The progress of the retinitis pigmentosa can be watched in the grafted eyes from its very beginning. The conclusions drawn from the facts presented are the following: retinitis pigmentosa is the cause of a disease; it follows the degeneration of the retina. Since the migrating pigment epithelial cells tend to remove the products of degeneration by their phagocytic action, it is a defensive mechanism against the disturbance. In no case, neither in the grafted eye nor in fragments of tissue transferred to a culture medium was there ever observed a migration of the pigment epithelial cells except the pigment epithelium was freed from the adjoining layer of the retina, either by degeneration of this layer or by detachment of the retina. It seems, then, that the pigment epithelial cells must be freed from the rods and cones; otherwise they are not able to migrate.

The facts mentioned in this paper were illustrated by twenty-two lantern slides and by a living specimen of the American tiger salamander (*Amblystoma tigrinum*) to which an eye had been grafted from another animal of the same species eight months before demonstration; the eye was in a normal condition so far as could be judged from its appearance.

## THE OPERATIVE TREATMENT OF TRAUMATIC COLOBOMA OF THE LID.

BY PROFESSOR L. v. BLASKOVICS, BUDAPEST.

Translated from *Arch. f. Augenheilk.*, vol. lxxxi., 1916.

(*With five figures in the text.*)

THE most frequent war injury to the eyelids is the coloboma of the eyelid. The cases usually come to us in the cicatricial stage. While these injuries occur during peace times, they are so uncommon that no typical operation has been developed. The principle for the reconstruction of the eyelid has remained unchanged and consists in reproducing that condition which was present directly after the injury and then to suture the wound in such a way that the lid returns as much as possible to its normal position. This principle cannot always be obtained, as the injuries consist not only in tears of the lids but often defects which must be replaced by fresh tissue. Hence the operative procedure is necessarily an atypical one. Nevertheless the frequency of these cases in war times has developed definite types of operation which more or less follow the two following varieties:

I. In these cases the eyelid is torn away from the canthus, usually the inner one. A scar is present which runs downward from the angle of the lids, varying in length. The lid is anchored fast in an ectropionated position. The difficulty of suturing the eyelid to its original position is given by the absence of a sufficient raw surface at the canthus whereby a proper union of the wound edges and a satisfactory adhesion of the eyelid in its original position can be obtained. Under these conditions the eyelid, though it is in its normal position

at the conclusion of the operation, through renewed contraction the ectropion returns. In order to correct this defect, it has seemed advisable to fasten the lid to the canthus in a way which can be described as hanging the lid upon a small flap or spur.

I find that von Pflugk has designed a similar procedure and has obtained good results with it. The method is extremely simple and consists of the following steps:

(a) The scar is outlined by two parallel running incisions which are joined at their extremities. After thoroughly excis-



FIG. 1.

ing the scar tissue as well as the lateral strands, the lower lid is perfectly free.

(b) At the inner margin of the wound a flap is formed with its base at the level of the angle of the lids (Fig. 1).

(c) At the temporal margin of the wound just below the adherent margin of the lid a straight incision is made into the skin. This small incision gapes in the form of a triangle and serves to receive the little flap which has just been described in *b*.

(d) The wound edges are carefully united (Fig. 2). The little spur-like flap fits smoothly into the triangular gaping wound just described. The remainder of the wound can be sutured with a continuous suture. The recovery in my cases has been unusually smooth. The new adhesion of the lid was

always sufficiently strong and the spur-like flap gave a large enough raw surface to unite the lid to. The spur itself becomes a part of the skin of the lid. No relapse whatever has been observed.

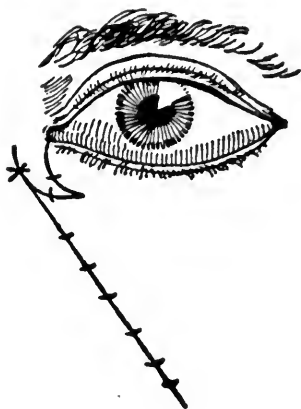


FIG. 2.

This operation is as well suited to the upper lid as to the lower.

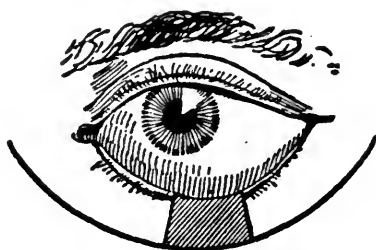


FIG. 3.

II. The second procedure is suited for those uncommon colobomata of the lower lid in which the margin of the lid has suffered traumatism in its middle part. After cicatrization of these wounds at the line of separation of the lids there is a cicatricial area between the two margins of the lid and the lid itself is ectropionated.

(a) The first incision is made at about 1cm below and parallel with the margin of the lid and throughout the whole length of the lid (Fig. 3).



(b) The scar is then circumscribed with two incisions which run vertical to the margin of the lid and completely separate the bridging flap.

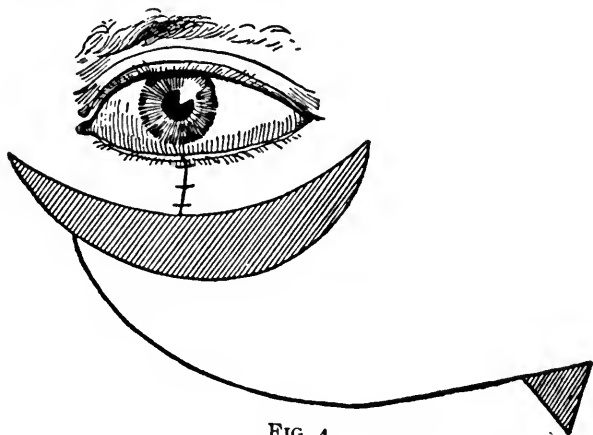


FIG. 4.

(c) The scar is thoroughly dissected and its lateral expansions are excised.

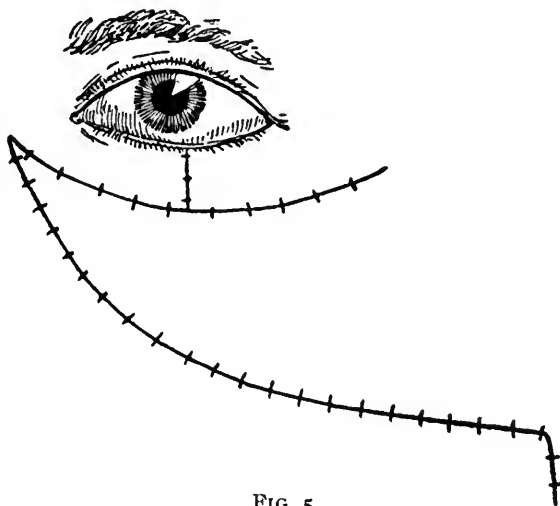


FIG. 5.

(d) The margins of the coloboma are then approximated by three interrupted sutures. The ectropion is thereby corrected and a semi-lunar raw surface is formed underneath the sutured lateral flap (Fig. 4).

(e) To cover this wound surface another curved incision is made running from the lower margin toward the temple. The apex of this new-formed flap is  $1\frac{1}{2}$  to  $2\text{cm}$  to the temporal side of the inner canthus. The lower extremity is considerably further out than the level of the outer canthus.

(f) After detaching this flap a triangular piece of skin and subcutaneous fat is excised from its lower margin. The base is directed up and measures  $1\frac{1}{2}\text{cm}$ .

(g) The columns of this triangle are first united. The flap is thereby displaced toward the nose and covers the defect by occupying a broader surface of raw area than before. The flap is then sutured with a continuous suture (Fig. 5). The results of this procedure were just as good as the first one. The flaps unite, owing to their broad base, without any trouble. The position of the lid is normal. Relapses were not observed.

## OPTICO-CILIARY NEUROTOMY, RESECTION OF THE OPTIC NERVE. A SUBSTITUTE FOR ENUCLEATION.

By DR. A. H. PAGENSTECHE, WIESBADEN.

Translated from *Archiv f. Augenheilkunde*, vol. lxxix., 1915.

**T**HIS operation, which is of course an old one, has in recent years been performed quite frequently at our hospital. It results in furnishing a good stump for a moveable glass eye, in case the latter is at all necessary. From 1898 to 1913 this operation has been performed more than 100 times in our eye hospital. In most of these cases the indication was not to shield the eye from the danger of sympathetic inflammation, but to preserve the blind eye if it was entirely free from danger to the other eye, from the standpoint that a blind eye was always better than an artificial one.

This resection of the optic nerve is indicated in my opinion in two groups of cases. The first are the cases of absolute glaucoma with great pain. The second those of total staphyloma where the staphylomatous formation has not advanced to the point of causing a great deformity. In my experience resection is followed by a reduction of the intraocular tension, the pain in the glaucomatous eye is relieved, the glaucomatous process in the eye with the staphyloma is arrested. An artificial eye is usually not necessary. If the patient for cosmetic reasons desires an improvement in his appearance, a thin glass shell can be made to fit over the affected eye, thus giving much better cosmetic results than with an ordinary artificial eye. The patient moreover is not required to wear his artificial eye constantly.

A few other cases remain of eye injury where the danger of

sympathetic inflammation could not be definitely excluded, but where enucleation was not concurred in. The resection is then practiced of a large piece of the optic nerve, believing that hereby in many cases a definite prevention of sympathetic inflammation is obtained. The eyeball after this operation shrinks, particularly if the injury has been a penetrating one, and in all circumstances forms an excellent cushion for the artificial eye. In my experience resection of the optic nerve in absolute glaucoma or in total staphyloma has never led to an atrophy of the eyeball, as is believed by many.

Our method of operating is as follows, which agrees entirely with the method as first advocated by my father, except that the resection of the muscles is no longer thought necessary. The conjunctiva is divided between the inferior and internal recti muscles about 1 cm distant from the corneal limbus. The scissors then proceeds into the deeper parts along the sclera. With a double hook introduced in the scleral tissue posterior to the equator in a meridian which connects the corneal diameter with the optic nerve entrance, the eye is drawn upwards and outwards, keeping traction exactly in this meridian. This puts the optic nerve on stretch and with strong scissors curved on the flat, the optic nerve is divided well back of its insertion into the eyeball. Keeping up the traction with the hook, the optic nerve remaining attached to the eyeball is then brought forward on the flat of the scissors until it appears in the wound. The optic nerve is then seized with a toothed forceps and the nerve is divided close to the sclera. All of the ciliary nerves about the optic nerve entrance are then divided. The eyeball is allowed to return to its normal position and it is very important at this moment to begin to exercise strong pressure upon the eyeball in order to prevent a retroocular hemorrhage. The conjunctival wound is closed with one or two sutures and a firm pressure bandage applied. The after treatment is not longer than the one following simple enucleation, if no pronounced bleeding has taken place.

I hope this short description of the operation will correct the impression, which seems to be general, that this operation is a difficult one, and in the near future I shall subject the cases which have been operated on by this method to a careful reëxamination, which I shall then be glad to publish.

## REVIEW

R. FOSTER MOORE. **The Retinitis of Arteriosclerosis, and its Relation to Renal Retinitis and to Cerebral Vascular Disease.** *Quarterly Journal of Medicine*, vol. 10, nos. 37 and 38, October, 1916, January, 1917.

The one hundred and ten cases which form the material for this study were examined and kept under observation as long as was possible at a general hospital and at an eye hospital. The cases were all subjects of general arteriosclerosis and were divided into two groups: (1) those who came to the eye hospital (Moorfields) because of impairment of sight due to vascular disease which had involved the retina, and (2) another group of patients at a general hospital (St. Bartholomew's) who were suffering from the results of cerebro-vascular disease and in whom the eye changes had given rise to no symptoms.

The author confirms the signs of retinal arteriosclerosis which were first pointed out by Marcus Gunn, and believes that obstruction of the blood-flow in the veins where they are crossed by the arteries is worthy of special emphasis, and is the most valuable and important single sign of severe arteriosclerosis. This phenomenon at the arterio-venous crossings is never seen apart from arteriosclerosis and is always present in sclerosis of any considerable degree. The variations in the degree form a reliable means of judging the extent of the sclerosis. The author has drawn attention to the displacement of the lines of the vein at these crossings which he thinks is a very striking and important symptom. He considers this phenomenon under two heads: The evidence of obstruction to the flow in the vein and displacement of the line of the vein. The distension which a vein peripheric to the crossing shows is generally not marked and is not an indication of the degree of obstruction. When a vein is

crossed twice by the same artery the trapped portion of the vein is very much reduced in size and when a vein crosses over a sclerosed artery an actual narrowing of its lumen can be seen. If an artery crosses a vein at an angle in the early stages of sclerosis the line of the vein will remain unaltered; as the thickening of the arterial wall increases, the line becomes diverted in proportion to the degree of the sclerosis. In other words, as a vein and an artery converge, the vein at first is diverted so as to lie alongside of the artery; it then passes under the artery and on the other side again runs alongside of the artery for a short course. This same phenomenon the author points out occurs when a vein crosses over an artery.

Retinal hemorrhages, the author believes, are directly due to the impairment in the nutrition of the vessel wall and to changes in the blood. The hemorrhages are not necessarily related to hyperpiesis on account of the following reasons: (1) pressure in the retinal arteries is not increased in arteriosclerosis; (2) a marked increase in the number of hemorrhages is not infrequent before death when there is no increase of blood pressure; (3) the retinal hemorrhages are very frequent in the various anemias in which there surely is no increase of blood pressure.

Under the heading of Arterial Disease as a Cause of Optic Atrophy, the author states that atrophy of the optic nerve either partial or complete may be brought about in two ways as a result of arteriosclerosis: first by thrombosis of a diseased artery with sudden loss of sight and subsequent optic atrophy, or the sclerosis of the arteries may slowly reach such a stage that they are no longer able to transmit sufficient blood to the tissues. This latter group is a particularly interesting one. The histories of eight patients are given, in whom partial atrophy followed from this cause. They were all the subjects of severe arteriosclerosis with marked disease of the retinal vessels. The loss of sight gradually developed in the course of several weeks or months. To the ophthalmoscope disease of the retinal arteries is well-marked; there may also be a little œdema of the disk. The fields show irregular constriction. The author agrees with Nettleship and Gunn, that retinal arteriosclerosis is more frequent in the female sex.

A very important and interesting part of this article is devoted to the discussion of Arteriosclerotic Retinitis as a clinical entity. In this, retinal exudates occur in eyes with retinal arteriosclerosis differing in appearance from renal retinitis and sufficiently distinctive for the author to set up a new disease, arteriosclerotic retinitis. In this condition there is disease of the small vessels. There are well-marked signs of retinal arteriosclerosis and retinal hemorrhages are present. The retinal exudates which are characteristic occur in the form of small white dots, spots, or small areas. They are irregularly circular in outline, vary in size, not larger generally than the diameter of a retinal vein. There is no oedema and no pigmentary disturbance. The dots sometimes seem to be in relation to the branches of the smallest veins, in other cases they have no definite relationship. It is common to find a group of them between the macula and the disk, and sometimes they constitute a complete or a partial star-figure around the macula. These dots are slow to develop and are very slow to undergo any changes, though the author has observed that they may completely disappear without leaving any trace. In the advanced cases there are some larger plaques which are identical with these discrete dots and never present any oedema, hemorrhage, or pigmentary changes. The arteries sometimes show a white plaque-like deposit in their perivascular sheaths. This is entirely different from the ordinary sheathing of the vessels, as the blood stream is not affected and the plaques may completely disappear.

Arteriosclerotic retinitis is often present in only one eye. A characteristic is the scantiness of the white dots, their arrangement, their course, and that they are always associated with severe arteriosclerosis of the retinal vessels. The usual exudates and hemorrhages seen in renal and diabetic retinitis are always absent. The diagnosis as to life in these cases is very uncertain, as they often live a number of years. The cause of death then is a disease of the vascular system and not one of the kidney.

A. K.

REPORT OF THE PROCEEDINGS OF THE SECTION  
ON OPHTHALMOLOGY OF THE NEW YORK  
ACADEMY OF MEDICINE.

By JOHN M. WHEELER, M.D., SECRETARY.

MONDAY EVENING, FEBRUARY 18, 1918. MARTIN COHEN, MD., CHAIRMAN.

Dr. M. URIBE-TRONCOSO presented a case of **chorioretinitis with fixed opacities in the vitreous**. A man 35 years old had a marked chorioretinitis in the left eye. The usual symptoms of cloudiness of the vitreous, large floating opacities, some exudates on the retina, papilla congested, and hazy and tortuous veins were manifest, but on looking carefully with direct ophthalmoscope and +7. or +8. in the sight hole, a curious aspect was detected. In the middle part of the vitreous, nearer the upper and inner side, appeared numerous small, gray-black, round, sharply outlined specks, mostly about 1mm in diameter, diffusely distributed and sometimes arranged geometrically in squares or constellations. By shifting the mirror in such a manner that only the edge of the beam of light falls upon the specks, they take a whitish transparent color with darker centers. These specks are stationary, as can be proved by focusing one group and having the patient's eye moved up and down and again fix. They are studded in one membrane, whose edge is visible on one side, and keep their arrangement and reciprocal distance on all movements. The anterior segment of the eye is normal. Vision  $\frac{20}{100}$ . In the right eye vision  $\frac{20}{30}$ . The fundus looks at first glance normal, but the vitreous is slightly hazy. With +7. a group of specks similar to the other eye, but less



numerous and of lighter color, can be detected. They are also fixed, but no sign of membrane or liquefaction of the vitreous could be observed.

The significance of these fixed opacities is interesting. Straub and Dhali have mentioned similar appearances which they call fixed vitreous dust and compare to the deposits on Descemet's membrane. They think they are heaps of leucocytes strewn on a membrane, which always form the wall of a cavity containing liquefied vitreous. The single leucocytes being too small cannot be detected by the ophthalmoscope and only produce a light haziness of the vitreous. They remain always isolated in the substance of the vitreous, never forming groups, which are only present on the walls of the cavities. The dust has been considered for a long time as a characteristic symptom of syphilitic chorioretinitis, still Dhali in fourteen cases found only one of true syphilitic origin and eight of tuberculous.

In Dr. Uribe-Troncoso's case although the left eye had certainly a liquefied vitreous and a membrane existed in which the heaps of leucocytes were fixed, in the right eye the condition seems different because the specks are not studded on a membrane on the wall of a cavity, as Straub believes, but were probably fixed by the coagulation of the fibrin in the serous exudate produced by the beginning diffuse inflammation. The question may also arise if these opacities do not represent the proliferation of connective tissue cells of the fibrillary stroma of the vitreous, made apparent by the inflammation on the knobs of the mesh; still its appearance is highly suggestive of heaps of leucocytes, similar to deposits on Descemet's membrane.

Dr. J. W. WHITE presented a case of **spasm of accommodation** (a full report appears in this issue).

Dr. WHITE showed also a case of **obliterating endarteritis**, probably tuberculous in origin. L. N., aged 20, developed sudden blindness in left eye over night two months ago. No history of illness. Vision R.  $\frac{20}{16}$ ; L. eccentric vision only, confined to a sector in the upper field. The fundus showed an obliterating endarteritis of the superior temporal and nasal vessels, the retina in the corresponding part of the eye ground showed deep opacities and hemorrhages. Wassermann nega-

tive. Tuberculin tests gave a slight febrile reaction but a decided focal reaction in fundus.

Dr. DUANE presented for Dr. S. T. HUBBARD a case showing **congenital retraction movements**. The patient is a Hebrew girl 16 years of age. She tilts her head to the left. When she attempts to look to the left the left eye shows limited abduction. When she attempts to look to the right (adduction) the left eye turns up and rotates inward, and it recedes into the orbit, allowing narrowing of the palpebral fissure. The patient has myopic astigmatism, but the vision is normal in each eye with the correction.

DISCUSSION. Dr. JULIUS WOLFF: I am interested in Dr. Hubbard's case of retraction movements of the eyeball because I was the first in this country to describe the clinical entity or syndrome of which the retraction is one symptom. My article appeared in the ARCHIVES OF OPHTHALMOLOGY, May, 1900. I here reported and analyzed five cases and emphasized the opinion that these cases were not so very infrequent but that the retraction of the eyeball had been overlooked. Dr. Herman Knapp told me at the time my cases were presented at this Section that he had not seen a case before, but in the above number of the ARCHIVES he also published a case.

If I may read the summary of my article it will be found to cover all the points brought out by Dr. Duane this evening.

"Retraction movements in the human eye have been described in only seven cases, to which five are herewith added. The retraction movements never occur as a solitary symptom, but always form part of the same group of clinical symptoms, producing a well-defined clinical picture, whose characteristics are as follows: The condition is always congenital. Retraction occurs during attempted adduction, which may be absent or present, but is always less than normal. Retraction is accompanied by narrowing of the palpebral fissure. Partial or complete paralysis of the external rectus of the retracted eye is regularly present. Some cases present a moderate constant retraction and narrowing of the fissure even in the primary position. In these cases attempts at abduction produce a propulsion of the globe and widening of the fissure. When the retraction is considerable the cornea is turned upwards in some cases, downwards in others, even when the fellow

eye makes a purely lateral movement. This is due probably to resistance made by the optic nerve.

"Two explanations are offered to account for the retraction: the *faulty insertion* and the *fixation* theories. Though the former is supported by some evidence, the latter accounts more satisfactorily for all the symptoms and is even better supported by evidence. Surgical interference may benefit some of the cases. There is reason to believe that the retraction movements are often overlooked and that these cases are not so rare as the small number reported would indicate."

Dr. Wolff showed a model illustrating the mechanism by which the retraction is brought about.

Dr. ARTHUR J. HERZIG presented a case of **organization of lens matter in the capsule**, the center having been absorbed. This shows as a dense annular ring, creamy white in color and apparently well organized, situated behind the iris in the position of the capsule. The patient, age 39, white, was struck in the left eye by a stone at the age of twelve. Iridectomy was performed six months afterward. The eye never gave the patient any trouble until December 27, 1917—twenty-seven years afterward, when he fell downstairs striking his eye with the handle of a coal scuttle which he was carrying at the time. The eye became inflamed and only local remedies were applied at the time until the eye became painful and he presented himself at Dr. Cutler's clinic at St. Luke's Hospital where I first saw him. Wassermann and Von Pirquet tests are negative. The eye is only slightly painful and all inflammatory signs have disappeared. Vision is nil in the injured eye, and normal in the other. As long as the eye is quiescent the speaker will not remove it. The case is presented to show the annular character of the organization of lens matter with the center of the lens absorbed.

Dr. GEO. H. BELL presented a case of **pulsating exophthalmos** due to a fracture at the base of the skull, in a man aged 46. Patient fell off a load of lumber about six weeks ago, striking on the back of his head. At that time patient bled from nose, mouth, and ears and was unconscious only a few minutes. Also he vomited some blood. Ten days ago he came to Dr. Bell's clinic at the New York Eye and Ear Infirmary complaining of headache, buzzing noises in ear and head. There was

much proptosis and marked restriction of the movements of the globe. When the eyeballs are pressed back into the orbit, you can see and feel the pulsation. Patient complains of diplopia. There is an audible bruit in both temporal regions, more pronounced on the right side. Pressure on common carotid on right side stops the bruit. There is an engorgement of the retinal veins in both eyes and some chorioretinitis in right. V. O. D.  $\frac{20}{50}$ , V. O. S.  $\frac{20}{40}$ . The congestion of conjunctiva has increased. The exophthalmos in right eye is 24 and in the left 22. X-ray plate showed diseased ethmoids and frontals, but no fracture could be made out. This is a case of an aneurism of internal carotid in the cavernous sinus. Blood pressure 120. Urine normal.

Dr. Bell expects to have a general surgeon ligate the common carotid on right side, and will make a report later on as to the result of the operation.

Dr. JOHN M. WHEELER presented a case of **paralysis of divergence**. The patient is a man (W. J.) 25 years old. August 1, 1917, while digging a ditch his vision suddenly became confused, and since that time he has seen double at a distance. He was in a hospital three months but left unimproved. His vision is normal in each eye, and there is no abnormal condition of the interior of either eye. Pupillary reactions are normal. The urine is normal. Wassermann tests of both blood and spinal fluid gave negative results. Neurological examination was negative. The characteristic features of divergence paralysis are well shown. The tropometer shows normal excursions of each eye in all directions. The far point for binocular single vision by approximation is about 14 inches, while the far point by recession is 24 inches. The point of equilibrium is 4 inches from the root of the nose. Diplopia decreases slightly as the patient looks to the right or to the left, whereas it would increase in looking in one of these directions if there were a paralysis of the external rectus. In this case paralysis of divergence probably resulted from hemorrhage into the divergence center, while the patient was under physical exertion.

DISCUSSION by Dr. CUTLER: I have seen two cases similar to the case reported. In one, a boy of ten, paralysis of the hypothetical center accompanied an attack of poliomyelitis

in which certain muscles of the neck and shoulders were involved. It was noticed as the boy recovered from the severe depression of the acute stage of the disease and remained stationary for a considerable time. The patient was taken to Boston later for treatment of the after-effects of the poliomyelitis, and I have not heard whether recovery of the function of the ocular muscles took place.

In another case, a woman with syphilis, the diplopia was typical and there was in addition a slight exophthalmos and tenderness on pressure of the left eye, as if there had been an involvement of orbital tissue, which I think was quite apart from the condition causing the diplopia, which was very characteristic of these cases. This symptom diminished very gradually under intensive treatment of the constitutional disease, but finally disappeared entirely.

In neither of these cases was the diplopia distressing. In the former the boy had considerable myopia and was able with his glasses quite readily to ignore one image or the other. In the second case there was moderate hypermetropia and the patient was more comfortable without glasses for distance.

In one other case at least, that I have seen, the diplopia was a distressing symptom and necessitated the closure of one eye, as prisms, base out, were not a satisfactory relief.

DISCUSSION by Dr. ALGER: Dr. Wheeler's case seems an absolutely typical one of divergence paralysis. My attention was first attracted to the condition by Dr. Duane's original paper to which reference has been made. Shortly afterward my first patient, a very intelligent gentleman, appeared and his description of his symptoms was enough to suggest the diagnosis. From time to time I have seen others, and in the *Transactions* of the American Ophthalmological Society of 1916 reported nine original cases.

I do not believe the condition can be anything like as rare as the very small number of reported cases would imply. The diagnosis is easy enough. It is simply overlooked because there is no reference to the subject in most neurological or ophthalmological text-books, and most of us do not think of the possibility of an ocular paralysis without limitation of rotation.

Cases like this would predicate the existence of a special

center for divergence, though so far as I know it has not been localized. Granting the presence of centers for divergence and convergence, on clinical grounds, there must, theoretically at least, be others for maintaining single vision in the other associated positions of the eyes. So far illustrative cases have not appeared.

The etiology, course, and prognosis seem to be much like those of the better known paralyses. My cases included probable hemorrhage from advanced angiosclerosis, syphilis, tabes; while one followed diphtheria, and was associated with paralysis of accommodation and deglutition.

Dr. EDUARD UHLENHUTH: **The influence of function on the structure of the eye** (an abstract of this paper is published in this number of the ARCHIVES). The paper was illustrated by many lantern slides and by a living specimen of the American tiger salamander.

MONDAY EVENING, MARCH 18, 1918. MARTIN COHEN, M.D., CHAIRMAN.

Dr. H. H. TYSON reported a case of **electric ophthalmia with chorioretinitis** which had been shown but not reported at the February meeting. "P. S.," age 53 years, visited the Knapp Memorial Eye Clinic on February 12, 1918, stating that his eyes burned and itched with some mucous discharge from the lids following an exposure to an electric flash of 670 volts, which occurred from a short circuit while fixing a motor the night previous. He stated that he had red vision for about ten minutes during which time he was practically blind. Examination of the eyes showed circumcorneal injection of both eyes, more marked in the right eye, with photophobia and lacrimation. Vision R.  $\frac{20}{100}$  and L.  $\frac{20}{70}$ . There was a central scotoma for red and green and metamorphopsia. Fundus right eye showed small oedematous patch in retina with two small linear hemorrhages located about two disk diameters to nasal side of optic disk (direct method) and in the left eye a patch of pigment in the choroid located somewhat in a similar position, as if being the result of previous exposure. The ophthalmoscope findings were similar to those found after direct exposure to sun in viewing solar eclipses. Vision improved later to  $\frac{20}{30}$  with correction. Up to 1913

only one case of marked chorioretinitis from the effect of electric light had been published, and that one was by Uhthoff. In May, 1913 (*Zeitschr. f. Augenheilk.*), Paul Knapp reported a second case. A man who had been exposed to a short circuit had vision reduced to one half and had metamorphopsia which later disappeared, and vision improved to normal. P. Knapp agrees with Fuchs as to the cause in these cases being the luminous rays, because they affect chiefly the choroid, pigment epithelium, and exterior layers of the retina, while the ultraviolet rays injure mostly the inner retinal layers. This case added to the literature makes the total three with chorioretinal changes.

The case was discussed by Dr. DENIG who had seen a similar case in which there was typical orbital neuralgia from short circuit.

Dr. A. D. MITTENDORF said that he had seen a case of chorioretinitis from bright lights in the Arctic region.

Dr. L. W. CRIGLER presented a case of **chronic simple glaucoma** in a young man, age 21, with a hypermetropia of 12 D. His father and mother are first cousins. They are living and healthy and have good vision. There is marked contraction of the visual fields. Vision is reduced to  $\frac{20}{200}$  in the right eye, and less than  $\frac{20}{200}$  in the left. The fundus showed the usual picture of chronic glaucoma. Tension in right and left eyes, between 30 and 35mm of Hg.

Dr. CRIGLER presented a case of **aphakia in a man 48 years of age**. The patient gives a reliable history of having had a cataract in his left eye twelve years ago. He does not recall having had an injury. On examination the eye presented the typical signs of a dislocated lens: the anterior chamber was deep, the iris was tremulous, the pupil was round, somewhat larger than that of its fellow, and responded feebly to light. Intraocular tension measured 30mm of mercury. Vision was reduced to hand movements at three feet in a very much contracted temporal field. The fundus could be best seen with a +8 D. lens. There were numerous opacities in the vitreous but the lens, even with the pupil dilated, could not be found. The optic nerve head was cupped and atrophic. The retinal vessels were pushed to the nasal side of the disk. Following the development of the cataract, the lens evidently

became spontaneously dislocated, associated with a tear in its capsular envelope, which in turn produced a secondary glaucoma. Except for the floating opacities in the vitreous the fundus could be easily seen and no evidence of the lens or its nucleus could be found. The right eye is normal.

Dr. GEO. H. BELL presented a case of **bilateral papillo-œdema** (published in full in this issue).

Dr. BRUDER thinks that papillo-œdema in this case was due to kidney disease and not to sinus trouble. Œdema, hemorrhages, and headaches might be caused by nephritis.

Dr. LESZYNSKY does not think that the patient presented symptoms which should have led to the diagnosis of tumor. He related a case presenting headaches, vomiting, and choked disk. Nephritis was present, and recovery ensued. He thinks that papillo-œdema of this type must be unusual in sinusitis.

Dr. GUTTMAN thought that it was unusual for papillo-œdema due to accessory sinus infection to be bilateral. Nephritis may have been caused by infection of the sinuses.

Dr. LAMBERT thinks that the neurologist should have been informed of the presence of nephritis. Recovery from albuminuric retinitis is common. He thinks it impossible to tell the cause from the ophthalmic appearance in papillitis.

Dr. LEVITT thinks that papillo-œdema in this case was not due to sinus disease but to nephritis.

Dr. ARTHUR S. TENNER presented a case of **chancre of the palpebral conjunctiva**. The patient is a young colored woman 22 years of age, married last December. She came to the clinic at the Harlem Hospital, on March 18th, complaining of a painless swelling of the left upper eyelid of two weeks' duration. There is a moderate circumscribed swelling of the lid producing ptosis. The swelling is indurated and extends over the whole upper lid. Neither the skin of the lid nor the lid margin has broken down. The eye is not irritated. On everting the upper lid the tarsus and tarsal conjunctiva are noted to be thickened and reddened with a sharply defined raised border. There are a few spots of dirty gray exudate. There is no pain nor tenderness. The preauricular gland is painlessly enlarged. A scraping of the conjunctiva showed numerous living spirochetæ. Some months ago this patient



had a "stye" in this eye. This stye may have been a chalazion which ruptured and left an opening in the conjunctiva for the entrance of the virus.

One week later a general maculopapular secondary eruption was present covering chest and abdomen and declared by a dermatologist to be of recent origin. The lesion of the lid was smaller and healthier looking and evidently in the terminal stage. No treatment had been used.

Dr. BRUDER said that it probably was not a chancre because the whole tarsus is involved, that it has not the characteristic ulcerated, punched-out appearance of chancre.

Dr. LAMBERT thought that both the appearance and the location argue against chancre. It may be tuberculous.

Dr. AGATSTON called attention to the history of a stye and thinks this may be an irritated chalazion.

Dr. E. M. ALGER presented a case of **pemphigus conjunctivæ**. This patient appeared at the Post-Graduate clinic a few days ago with the following history. She began having trouble with her eyes about three years ago which has steadily grown worse ever since in spite of constant treatment. The diagnosis was apparently made very early, both as to the eyes and the numerous lesions in the mouth, nose, and throat. She has never had any bullæ on the skin. She has, since that time, visited one hospital after another in the desperate hope of getting some relief. The left eye, which was first involved, was finally operated upon by one of our colleagues, who adopted the perfectly logical plan of attempting to prevent the cicatricial obliteration of the conjunctival sacs by extensive skin grafts. The result as we see it was complete failure. There is a complete symblepharon of both lids, while the margins of the lids are attached to each other throughout their entire length by a thick skin which completely covers the cornea, to which it is probably grown fast. The patient is rather bitter over the results of the procedure. As a matter of fact the operation had nothing to do with the final condition because the fellow eye gives every appearance of ending in exactly the same way at no very distant time. It shows a nearly complete obliteration of both conjunctival sacs, and the bridge of skin connecting the lid margins has already advanced far from the external canthus toward the cornea, only a small

part of which remains in sight. It is, however, so far, clear and the vision is good. Projecting into this space from beneath the upper lid margin is a large thick-walled vesicle similar to those on her lips.

Four types of pemphigus are recognized to-day. First, the so-called "essential shrinkage" of the conjunctiva, without vesicles; second, the cases in which lesions occur on the conjunctiva alone; third, cases with conjunctival and associated mucous lesions, and fourth, those associated with classical pemphigus of the skin. The difficulties of positive diagnosis in the first two classes do not need to be pointed out.

Pemphigus is one of the rare skin diseases, and conjunctival pemphigus is far more uncommon. The characteristic blebs may involve the outer layers of conjunctiva, in which case they are of short duration and rarely seen, or they may involve, as in this patient, the entire thickness and last several weeks. Corneal lesions are rarely seen in the vesicular stage. In any case the bullæ finally break down leaving raw surfaces which undergo extreme cicatricial contraction, or where they are opposed to each other, result in symblepharon and finally in obliteration of the conjunctival sacs and total blindness.

Nothing definite is known to-day regarding the cause of the disease. No successful method of treatment has so far been devised for real pemphigus. There is no natural tendency to get well; indeed, the prognosis is bad so far as life itself is concerned, while as to vision it is absolutely bad.

Dr. Denig saw this patient very early in the disease when the diagnosis was not so obvious. In fact he made it ahead of the nose and throat men which speaks well for his clinical acumen.

Dr. DENIG saw this patient about two years ago. She had purulent conjunctivitis and complained of sore throat. A rhinologist reported rhinitis. After treating her for six weeks Dr. Denig noticed adhesions in the lower fornix. He then discovered blebs in the mouth and nose. Adhesions increased and skin grafting was advised. Patient then disappeared.

Dr. LAMBERT saw a case of acute pemphigus with Dr. Huddleson several years ago. It followed submucous resection of the nasal septum and the appearance suggested small-pox. The patient died in four days of general septicæmia.

Dr. CURTIN saw a case of bilateral symblepharon three years ago and thought it trachoma. Later the patient died of pemphigus.

Dr. COHEN said that microscopical examination of the tissue in these cases early shows endarteritis, and later connective tissue cells.

Dr. MAX TALMEY read a paper on **suggestions for improving the cataract operation.** Homer Smith's preliminary capsulotomy with the author's modification always yields a large capsular opening. The latter and thorough separation of the cortex during the interval render possible complete removal of the lens and thereby fulfil the first requirement for a perfect cataract operation, *i.e.*, the attainment of entirely clear media. To obtain the second requirement, a round central pupil, there is only one way that can always be depended upon, and this is to close the wound completely by sutures. This has been tried extensively but all procedures hitherto devised have been imperfect and have caused frequent failure. The author's improved procedure consists in a downward section following capsulotomy after an interval of six hours. Three threads are inserted through the borders of the wound and then the lens is expressed. The middle thread is now tied even if the iris should happen to lie in the wound. After putting the iris in correct position the lateral threads are tied. The sewing of the wound differs essentially from that employed by other surgeons by the number of sutures, the inclusion of a sufficient amount of tissue, and the operative stage at which the threads are inserted. The author believes that these steps do not complicate the cataract extraction but rather facilitate it by rendering it more easy to deal with complications such as prolapse of iris and of vitreous body. This method with omission of capsulotomy would even divest the intracapsular extraction, but it was just the shortcoming of the latter that suggested the above operation. The danger of infection is decidedly enhanced by the sutures and must be obviated by the most scrupulous asepsis and antisepsis.

Dr. LAMBERT said that suturing entails risk to the eye. He thinks he should not feel justified in trying such a difficult feat. He thinks the Homer Smith operation accompanied

with danger, and has had glaucoma following preliminary capsulotomy.

Dr. TALMEY said that he had not had an opportunity to try the technique which his paper suggests but that his ideas were not less valuable because of being purely theoretical.

## REPORT ON THE PROGRESS OF OPHTHALMOLOGY FOR THE SECOND, THIRD, AND FOURTH QUARTERS, 1917.

By H. KOELLNER, Berlin; W. KRAUSS, Marburg; R. KÜMMELL, Erlangen;  
W. LOEHLEIN, Greifswald; H. MEYER, Brandenburg; W. NICOLAI,  
Berlin; H. PAGENSTECHE, Strassburg; K. WESSELY, Würzburg;  
and M. WOLFRUM, Leipsic, with the Assistance of Drs. ALLING, New  
Haven; CALDERARO, Rome; CAUSÉ, Mayence; CURRAN, Kansas City;  
DANIS, Brussels; GILBERT, Munich; GROENHOLM, Helsingfors; v.  
POPPEN, Petrograd; TREUTLER, Dresden; and VISSER, Amsterdam.

Edited by MATTHIAS LANCKTON FOSTER, M.D., F.A.C.S.,  
New Rochelle, N. Y.

(Continued)

### V.—ANATOMY, EMBRYOLOGY, MALFORMATIONS.

40. DAVIES, D. L. **Anophthalmos and microphthalmia.** *British Journal of Ophthalmology*, July, 1917.

41. MAGITOT, A. **The aqueous humor and its origin.** *Annales d'Oculistique*, cliv., 2, 3, and 4.

DAVIES (40, **Anophthalmos and microphthalmia**) contributes five cases of this anomaly to the literature on the subject. The possible causes are discussed in the paper, and three photographs illustrate the appearances of the patients.

T. HARRISON BUTLER.

MAGITOT (41, **The aqueous humor and its origin**) makes an exhaustive critical review of the entire literature on the subject, and comes to the following conclusions:

(1) The primitive aqueous humor is secreted during foetal life between the third and fifth month by the neuroglia cells accompanying the hyaloid vessels. These cells disappear after their secretory function has been fulfilled.

(2) The aqueous humor has a triple rôle: (a) optic and

static; (b) protective for the retinal elements; (c) as a conserving medium for the lens.

(3) Its chemical composition is very much similar to that of the cerebro-spinal fluid. It contains no colloids and no nutritive elements for the nervous tissue but it constitutes an excellent medium for the conservation of the energy of this tissue.

(4) Under normal conditions *the aqueous humor is without movement*. Contrary to the universally accepted theory, Magitot affirms that there is no circulating current in the anterior chamber and no drainage into Schlemm's canal. However it is probable, he says, that it is absorbed very slowly. Whatever quantity is absorbed is replaced by the neuroglia cells of the retina: the cells of Müller, those around the ora serrata, and the clear ciliary cells whose prolongations constitute the zonula. The fluid is produced by these cells by a process of dialysis, a phenomenon entirely different from secretion and transudation.

(5) There are two systems of lymphatics in the eye. One is independent of the other. They are to be found in the perivascular sheets. There is no direct communication between the aqueous humor and the lymph of the eyeball.

(6) The fluid reformed after a puncture of the anterior chamber is serum, transuded from the capillaries, and a slight amount of aqueous originating from the vitreous. The paper is a very able critical discussion of the theories advanced, experiments performed, and facts established mostly by Leber's school and contains a complete bibliography of great help to those who are especially interested in this subject.

SCHOENBERG.

#### VI.—THE SENSE OF SIGHT.

42. CERISE, L. Two cases of double hemianopsia with conservation of macular vision. *Archives d'ophtalmologie*, xxxv., 5.

43. EDRIDGE-GREEN, F. W. The relation of ophthalmology to the theory of vision. *British Journal of Ophthalmology*, July, 1917.

44. PARSONS, J. H. The apocritic principle and the evolution of visual perceptions. *Ibid.*, July, 1917.

45. TRAQUAIR, H. M. Bitemporal hemiopia: the later stages and the special features of the scotoma. *Ibid.*, March, 1917.

46. VINSONNEAU. The macular vision in hemianopsics with intact macula. *Archives d'ophtalmologie*, xxxv., 5.

PARSONS (44, **The apocritic principle and the evolution of visual perceptions**) describes the apocritic principle as a "schematic description of patent facts." It can be analyzed into two consecutive stages: a selective segregation, and a creative synthesis.

This paper does not lend itself to abstraction and should be read in the original.

T. HARRISON BUTLER.

EDRIDGE-GREEN'S (43, **The relation of ophthalmology to the theory of vision**) theory of vision may be stated as follows: A ray of light impinging upon the retina liberates the visual purple from the rods and a photograph is formed. The rods are concerned only with the formation and distribution of the visual purple, not with the conveyance of light impulses to the brain. The ends of the cones are stimulated by the photo-chemical decomposition of the visual purple by light, and a visual impulse is set up which is conveyed to the brain by the optic nerve. The character of the stimulus differs according to the wave length of the light causing it. But though the impulses differ in character according to the wave length of the light causing them, the retino-cerebral apparatus is not able to distinguish between the character of adjacent stimuli, not being sufficiently developed for the purpose. The power of discrimination differs in individuals, hence the varieties of color perception which are met with. The object of the paper is to show that every known aberration of function, in accordance with the theory, is represented by a disease of the eye, none of which have hitherto had a satisfactory explanation. Vision being photo-chemical there should be defects in accordance with well-known photo-chemical facts.

The rods having the special function of regulating the photo-chemical sensitiveness of the liquid surrounding the cones, there should be diseases corresponding to aberration of this function.

When the sclerotic, choroid, and the pigment cells of the retina are removed the external fovea is seen as a small basin with four canals leading to it. In normal conditions there is a circulation of lymph sensitized by the visual purple from the surrounding parts of the retina into the external fovea and then out through the choroid into the lymphatic spaces of the optic nerve. This retinal circulation is greatly aided by the

four canals. In cases of optic neuritis this starfish-like arrangements of the canals can be seen with the ophthalmoscope. It can also be seen in high myopia. In one case it was seen red and there was a small black spot at the center. Obstruction of the canal should cause night blindness, and this we find to be the case in myopia.

The paper goes on to consider the causation of nystagmus, amblyopia, erythropsia, night blindness, functional disturbances, reversal of the color fields, and scintillating scotomata.

T. HARRISON BUTLER.

VINSONNEAU (46, **The macular vision in hemianopsics with intact macula**) has noticed that patients with hemianopia due to a war injury of the head, with intact central vision and emetropia, have the acuity of vision reduced in the eye situated on the same side with the hemianopsia. In patients with right-sided homonymous hemianopsia the right eye is the poorer, and vice versa. The author called attention to this in 1915, and the fact has been confirmed by a number of other authors. In this paper he reports two more such cases.

SCHOENBERG.

TRAQUAIR'S (45, **Bitemporal hemiopia: the later stages and the special features of the scotoma**) paper runs through three numbers of the Journal. It is well illustrated, and should be consulted by neurologists and ophthalmologists specially interested in disease of the pituitary body, and chiasma.

The author sums up his conclusions as follows:

(1) The perimetric defects in bitemporal hemiopia follow a typical or normal development. Commencing in the upper-outer quadrant, the field is involved in a circular manner, the loss proceeding clock-wise in the right field, and counter clock-wise in the left.

(2) The central defect or scotoma behaves in the same manner.

(3) This type of field defect is due to interference with chiasmal fibers, but is largely independent of the ultimate cause; it occurs in bitemporal hemiopias from a variety of diseases.

(4) The immediate cause is probably a chiasmal neuritis comparable to that which, acting upon the optic nerve, produces a retrobulbar neuritis.



(5) The origin of this chiasmal neuritis is not known.

(6) In tumor cases, and perhaps in some non-tumor cases, mechanical pressure also acts, and mainly in the later stages.

(7) In these later stages the normal type of progress may be "swamped" by pressure effects and greatly altered.

(8) These observations provide evidence from the clinical side in support of the looped arrangement of the chiasmal fibers.

(9) They also show that the papillo-macular bundle forms a little chiasma within the chiasma, and that its fibers are similarly arranged.

T. HARRISON BUTLER.

CERISE (42, **Double hemianopsia with conservation of macular vision**) reports two cases of this nature. The first patient sustained an extensive war injury of the occipital region and, after the usual period of blindness, following such accidents, he recovered a vision of  $\frac{1}{10}$  O. D.;  $\frac{2}{10}$  O. S. The field, doubly hemianopic in each eye, was reduced to the central vision,  $10^{\circ}$  to  $15^{\circ}$  around the fixation point. He had also convergence insufficiency, hemiparesis, and difficulty in writing. The author believes that the lesion was bilateral and that an island of cortex in the right occipital lobe from which the fibers go to both maculae remained untouched. Since the crossed macular fibers are more numerous than the direct ones, the macula of the left eye is the better provided and the vision of this eye is better. The second patient, 74 years old and arterio-sclerotic, had for some time a right homonymous hemianopsia and set up, through some vascular changes, a left-sided hemianopsia. The macula remained intact. Vision O. D.  $\frac{1}{10}$ ; O. S.  $\frac{3}{10}$ . The field was reduced to  $10^{\circ}$  around the fixation point. According to the author an island comprising the macular center must have remained intact in the right occipital lobe which preserved the central vision in both eyes.

SCHOENBERG.

## VII.—REFRACTION AND ACCOMMODATION.

47. NORDENSON, J. W. **On the rules for the correction of astigmatism.** *British Journal of Ophthalmology*, March, 1917.

48. PATON, L. **Functional spasm of accommodation.** *Ibid.*, October, 1917.

49. POST, M. H. **Change of refraction following an attack of diabetes.** *American Journal of Ophthalmology*, November, 1917.

POST (49, **Change of refraction after diabetes**) observed the refraction in a diabetic woman after an acute attack and found an increase of  $+ 0.75$  D. Some months later the eyes returned to their former condition. No lens opacities were found.

ALLING.

The object of NORDENSON'S (47, **On the rules for the correction of astigmatism**) paper is to answer the question, whether in any given case astigmatism should be corrected or not.

*Indications for the correction of astigmatism.*

In general correction is needed only if it increases visual acuity. When correction improves acuity it may be advisable on account of the following factors:

- (1) The need of accurate detail vision.
- (2) Asthenopia: which is of two kinds:
  - (a) Accommodation asthenopia.
  - (b) Eyelid asthenopia. The orbicularis can correct as much as two to three diopters of astigmatism by pressure on the globe when the error is against the rule, but this action causes fatigue.

(3) Dangers to the eye from astigmatism. Astigmatism may favor the development of strabismus and myopia.

*Contraindications to correction.*

(1) The inconvenience of wearing glasses. If one eye be normal and the other astigmatic it is not generally necessary to order glasses. The exceptions are those cases in which there is asthenopia, or if strabismus is threatening. It may also be useful to correct the error when accurate binocular vision is called for.

(2) Non-improvement of acuity by correction. In this case it is useless to correct, but the author goes on to state what every ophthalmologist must have observed, that the vision of an eye which at first shows no improvement may, after a lapse of time which may extend to months and years, begin to gain in acuity.

(3) *Intolerance of correction.* In many cases the patient will not wear the correction even if accurate. This is especially

noted in elderly people who have never worn glasses. In the case of these older patients it may be impossible to persuade them to persevere with the glasses which cause discomfort.

(4) The author notes that Lagrange is opposed to prescribing glasses for children. This surgeon considers that there is a decided tendency for astigmatism to cure itself, and that this change is inhibited by wearing spectacles. Nordenson regards this hypothesis as unproved. The views advanced by the author will meet with little response in England or America, where the tendency to order an absolutely accurate correction of ametropia is steadily gaining ground. Most ophthalmologists will agree that an uncorrected error of refraction, especially astigmatism, even when no symptoms are evident, must be a cause of an expenditure of brain energy which in the long run tells its tale. In general the cases which do not require correction are those which never consult the oculist.

T. HARRISON BUTLER.

True spasm of accommodation is one of the rarest conditions met with in ophthalmology. Hirschberg, writing in 1884, says: "I scarcely believe in the existence of acute and not at all in the chronic form of accommodative spasm." Hess expresses similar views. True spasm of accommodation may be defined as the sudden development of a high degree of apparent myopia which disappears under atropine. PATON (48, **Functional spasm of accommodation**) cites a case of this nature.

T. HARRISON BUTLER.

#### VIII.—THE MOTOR APPARATUS.

50. CAMILLE and FROMAGET, H. Latent nystagmus. *Annales d'oculistique*, cliii., 11.

51. LEAVITT, M. J. A case of recurrent paralysis of the right abducens lasting twenty-one years. *Ophthalmic Record*, September, 1917.

52. MARDELLIS, A. Paralysis of one abducens due to a fracture of the petrous portion of the temporal bone. Recovery. *Clinique ophthalmologique*, viii., 3.

53. MENACHO, M. Associated movements of the upper lid during mastication (the Marcus Gunn symptom). *Annales d'oculistique*, cliv., 4.

54. ROUSSEAU, F. The paralysis of the oculomotor nerves during otitis media suppurativa and their complications. *Ibid.*, cliii., 12.

55. SUKER, G. F. A new ocular muscle symptom in exophthalmic goiter. *Journal of the American Medical Association*, April 28, 1917.

56. WOODRUFF, H. W. Tendon transplantation of the eye muscles. *Ophthalmic Record*, November, 1917.

LEAVITT'S (51, **Recurrent paralysis of the right abducens lasting twenty-one years**) patient was a woman of 23, who had twenty-five or thirty attacks of paralysis of the external rectus of the right eye, the first coming on during an attack of measles when she was a year and a half old. The attacks lasted usually about four or five weeks while the mobility was normal during the intervals. The probable explanation seems to be that some vascular or oedematous changes take place at the nucleus.

ALLING.

MARDELLIS (52, **Paralysis of one abducens due to a fracture of the petrous portion of the temporal bone**) tells us of a patient who was struck on the head by a falling tree and immediately complained of a severe headache, epistaxis, and diplopia. Examination revealed a complete paralysis of the left sixth cranial nerve. The paralysis remained stationary for about four months, when it disappeared suddenly, and the motility of the left eye again became normal. The author then modified his diagnosis of a complete severance of the left abducens, due to a fracture of the tip of the petrous portion of the temporal bone, as he is of the opinion that a complete division of the nerve would not have recovered suddenly. He thinks that a hæmatoma was formed which pressed upon the abducens and induced a temporary paralysis, and that this disappeared as soon as the clot was absorbed.

SCHOENBERG.

ROUSSEAU (54, **Paralysis of the oculomotor nerves during otitis media suppurativa**) enumerates the various types of oculomotor paralysis that may occur during acute and chronic otitis media purulenta and summarizes them in the following manner: (1) Paralysis of the sixth nerve, in benign cases, may appear in aseptic serous meningitis, or limited osteitis of the tip of the petrous portion of the temporal bone. In malignant cases it may appear in abscess of the cerebellum, or extradural abscess at the apex of the petrous portion of the temporal bone. (2) Paralysis of the sixth and fifth nerves, the symptoms of Gradenigo, may be produced in benign cases by a slight osteitis of the apex; in malignant cases by an extradural

abscess, or localized meningitis. (3) Paralysis of the sixth and third nerves appears usually in thrombophlebitis of the cavernous sinus. (4) Paralysis of the third nerve in an abscess of the temporo-sphenoidal lobe. SCHOENBERG.

CAMILLE and FROMAGET (50, **Latent nystagmus**) again call our attention to a condition they have already described under the term "latent nystagmus" and give five more histories of such cases. The condition seems to be analogous to latent strabismus. They believe that just as there is a fusion center which prevents heterophoria from becoming manifest, there is a center, located in the cortex, which keeps the nystagmus in a latent condition. As soon as one of the eyes is excluded from vision the function of this center is interfered with and the nystagmus appears. In each of the five cases reported the patient had one bad eye, and when the good one was screened a nystagmus appeared in the other. SCHOENBERG.

MENACHO (53, **Associated movements of the upper lid during mastication**) reports a case in which the right upper lid was retracted every time the lower jaw moved downwards. He discusses the various possibilities of nuclear, subcortical, and cortical localization, as well as of an abnormal arrangement of the connecting fibers between the nuclei of the fifth and seventh nerves, and declares his inability to agree with any of the theories which have yet been advanced.

SCHOENBERG.

SUKER (55, **New ocular muscle symptom in exophthalmic goiter**) asserts that, after extreme lateral rotation of the eyes, when an attempt is made to rapidly fix an object in the median line, one of the eyes fails to follow the other or lags behind, but eventually jumps into place with the other.

ALLING.

A few attempts have been made in general surgery to transfer an active muscle to take up the action of a paralyzed one. WOODRUFF (56, **Tendon transplantation of the eye muscles**), following the technique of Hammelsheim, has operated on two cases of paralysis of the external rectus by dividing the superior and inferior recti tendons and suturing half of each to the tendon of the externus. The results seem to have been a slight improvement in the internal strabismus, but no restoration of motility outward.

ALLING.

## BOOK REVIEWS

V.—**Tests for Color Blindness.** By Professor SHINOBU ISHIHARA, Tokyo Handaya, 1917.

This series of plates is designed to discover quickly defects in color perception, particularly in railway employees, candidates for the navy, and others. The color effect which enters into consideration is always congenital and concerns red and green blindness. The plates are so designed as to apply the peculiarity of the red and green blind consisting in the fact that blue and yellow colors appear to them much brighter than the red and green colors. Each plate consists of a collection of round disks of pigment of varying diameter in which a central figure against an appropriate background will appear as a certain number to the normal person and as a different number to the abnormal individual. The tests are very ingeniously designed and undoubtedly furnish a valuable addition to our equipment for detecting color defects. The get-up of this little book is worthy of note and is a testimonial of the high standard of bookmaking in Japan.

A. K.

VI.—**Oral Roentgenology.** By Dr. KURT H. THOMA, Lectures on Oral Histology, Harvard Univ. Dental School. Pp. 213. 311 illustrations. Boston, Ritter & Co., 1917.

The importance of dental infection in eye disease requires some knowledge of the X-ray findings in normal and pathological tooth conditions. This book, a Roentgen study of the anatomy and pathology of the oral cavity, admirably furnishes this information. The normal oral tissues and their pathological changes are carefully described and illustrated by Roentgenograms. The author, however, correctly states that

the Roentgen examination does not replace all other means of diagnosis, but it should be used in conjunction with other well-recognized modes of examination. The book is a scientific contribution to an important subject and its practical value is increased by brief case reports and their X-ray findings.

A. K.

**VII.—Military Ophthalmic Surgery.** By Drs. ALLEN GREENWOOD, GEO. E. DE SCHWEINITZ, and WALTER R. PARKER. War Manual No. 3, pp. 115, illustrated. Lea & Fibiger, Phila. and New York, 1917. Price \$1.50.

This little book is designed for medical army officers who have to deal with special ophthalmic problems. The first part on Military Ophthalmic Surgery is written by Greenwood and is based on the results of experience gained at the war front in the British hospitals. This describes contusions and wounds of the eyeball, the implantation of a glass ball after enucleation, the value of the X-ray in localizing foreign bodies, the methods for their extraction, the use of conjunctival flaps, traumatic cataracts, wounds of the orbit and of the eyelids, and a brief reference to the fundus changes in trench nephritis and intracranial injuries. Finally, a list of the eye instruments for a base hospital is given. Trachoma and the Common Forms of Conjunctivitis are described by de Schweinitz, and Parker contributes a chapter on Malingering.

A. K.

**VIII.—The Stereoscope in Ophthalmology.** By Dr. DAVID W. WELLS, Boston, pp. 143. Published by Globe Optical Co., Boston, 1918.

The little book which appeared in part in 1912 deals principally with stereoscopic fusion training. The author recommends the phoro-optometer for this purpose which makes use of the principle of the insinuation of the prismatic element by decentering strong spheres, the objects remaining fixed. The stereoscopic treatment of exophoria, esophoria, hyperphoria, and heterotropia is then taken up in turn. A chapter is devoted to the musculo-capsular advancement operation and the author gives his modification of the Worth technic,

consisting in a so-called scleral anchor and the use of a fixation hook. Finally the Haitz method for the binocular examination for scotomata with the stereoscope is fully described; in this test the author prefers to use the phoropter.

A. K.



## ARCHIVES OF OPHTHALMOLOGY.

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### SOME OCULAR CONDITIONS AFFECTING THE EFFICIENCY OF THE AVIATOR.<sup>1</sup>

BY COLONEL WILLIAM H. WILMER, MEDICAL CORPS, N.A.

*(With six illustrations in the text.)*

THE medical problems involving the aviator are unique. The airman is compelled to spend much time in an unaccustomed environment. In addition to the stress of conflict common to all branches of military service, he is compelled to subject himself to sudden changes—so sudden that there is but little opportunity for adaptive adjustments to occur. There are sudden changes of temperature, barometric pressure, muscular and mental exertion, from ground to labile equilibrium, from normal sea-level oxygen tension to a tension much decreased.

Under conditions of lowered oxygen certain well-known physiological changes occur, which are adaptive in nature. There is a cry for help from the tissues of the body which demand more oxygen. The blood stream is not only a sewer carrying off waste products but a nutrient fluid and a carrier of oxygen. This appeal for succor is met in good subjects by an increase in depth of respiration, by the dilation of peripheral vessels, and by more rapid heart-beats. More frequent respiration and an increased blood pressure are clumsy attempts at adaptation, and the airman soon breaks

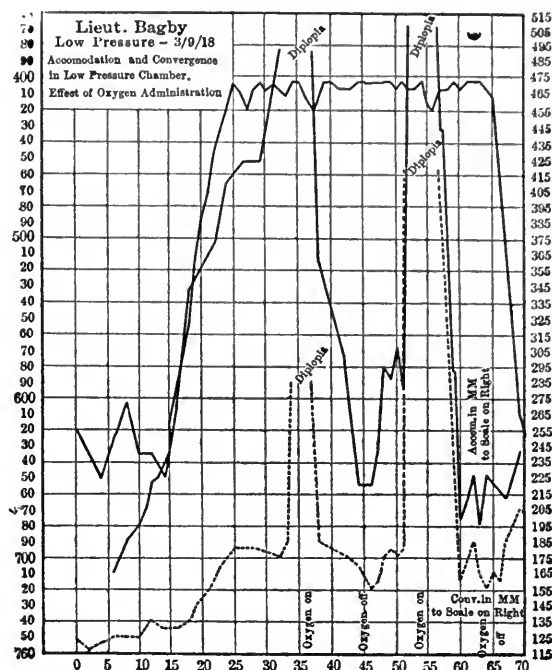
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<sup>1</sup>Read at meeting of American Ophthalmological Society, New London, July, 1918.

under such tension. In the blood itself, there are changes consisting of concentration and an actual increase of available red blood corpuscles.

At the Medical Research Laboratory, the conditions of lowered oxygen tension are produced artificially by two different forms of apparatus:

- (1) A large low-pressure chamber from which the air is



removed by an electrically driven pump, the oxygen tension and the barometric pressure being simultaneously lowered, while the amount of  $\text{CO}_2$  increase in the chamber is negligible, owing to the good ventilation.

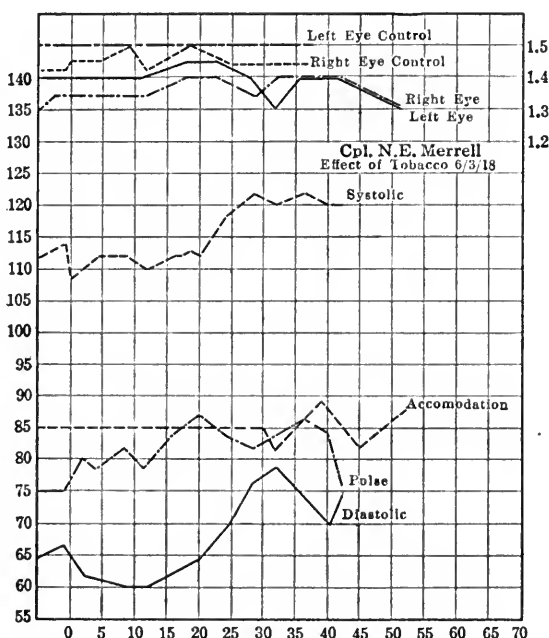
- (2) The Henderson rebreathing apparatus, where the subject breathes up his own oxygen from a tank, the excess of  $\text{CO}_2$  being absorbed by sodium hydroxide.

Tests made by these different forms of apparatus correlate so closely that we are compelled to believe that the physiological changes noted are due to the want of oxygen and not to

the lessened atmospheric pressure. This is further proved by the fact that the administration of oxygen causes a quick return of the disturbed functions to the normal.

It is difficult to say how much of the disturbance of function is due to suboxidization in the central nervous system and how much is due to changes in the muscles themselves. At all events, the results are strikingly similar to the toxic effect of alcohol, etc.

In various accident reports from aviation sources there



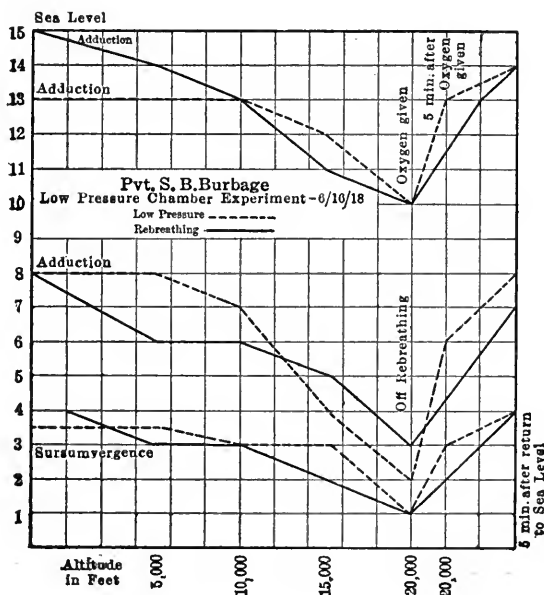
is constant reference to the statement by pilots that vision was blurred during flight. In many cases the instruments could not be read correctly, distance from the ground properly judged, etc. We have, therefore, been interested in making our tests under conditions of lowered oxygen pressure to see just what changes do occur in the various functions of the eye. It is well known that the visual requirements for the air service of the United States are much stricter than

those of any other nation, and there have not been many instances where careless selective examinations have passed candidates with defective vision or muscular imbalance. The fact that the ocular functions (especially the motor apparatus) do fall off in an atmosphere of reduced oxygen pressure convinces us of the continued necessity of high entrance requirements. The acuteness of vision, of course, is the most important ocular asset of the aviator. In testing this function under low oxygen conditions, a number of factors have to be considered, such as practice, suggestion, concentration, etc. On the whole, we have not found any *very marked* change in the acuteness of visual perception. The great variations have been due to the falling off in the power of muscular adjustment. For instance, in hyperopic individuals there is likely to be a deterioration owing to the reduction in the power of the ciliary muscle. On the other hand, in those slightly myopic, there is a tendency towards improvement, for the same reason. Our experimental and practical experiences have not borne out the statement of some of our confrères that vision in the air is improved at the height of about 200 meters, owing to a congestion of the head, the choroid, and the retina. To test this, we tried the effect of nitrite of amyl on the sight of a number of observers. There was falling off in vision in every instance except in one myopic subject, where there was improvement.

Normal color vision is most important for the aviator; but we have found very little practical change in this function though there have been found some alterations of its threshold values.

A large field of vision is very necessary for a good aviator. This is especially important because the fuselage causes such a large blind angle in the visual field. In the tests of the visual fields some rather definite changes have been noted, in some instances indicating that the periphery of the retina is much more sensitive to oxygen want than the macular region, either through the nervous apparatus of the retina itself or through suboxidization in the perceptive centers. While in the average we have not found any great change, in some cases up to 10,000 feet the visual fields seem to increase; about 15,000 feet there is a slight contraction; at 20,000 feet there is a marked contraction for form and color.

Retinal sensitivity is not tested in the original selection of the aviator; but later, in his classification, this test is essential, and it is also necessary to determine whether this function is diminished under certain air conditions. The test for threshold retinal sensitivity is interesting and, for laboratory purposes, a very practical and exact test. However, it requires too much time to be useful as a quick clinical test. So, in substitution for this, we use a comparative sensitivity test.

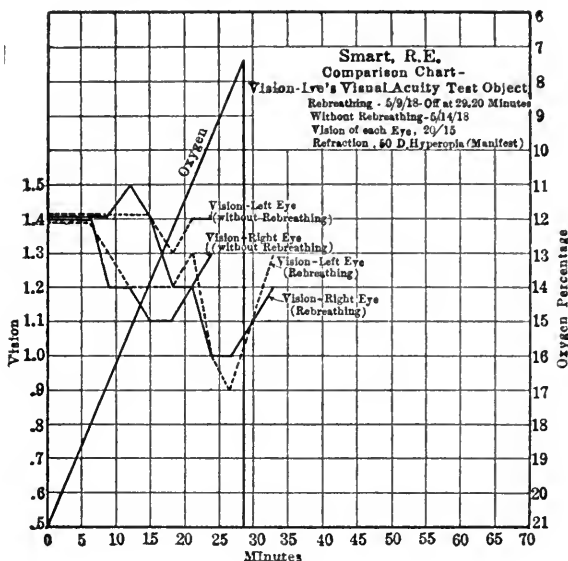


In this test the object is a gray letter on a lighter gray background, there being thirteen shades of perceptible difference between them. One eye is closed and the photometric wedge is passed before the other eye until the letter blends in to the background and can no longer be recognized. The point at which this occurs is noted on the millimeter scale of the wedge. The time allowed for the process is from five to eight seconds. It is repeated three times for each eye. The wedge that is used was devised by Lieutenant Reeves. It consists of a layer of gelatine of increasing density of color between two pieces of optical glass. The light transmission has

been calculated for each millimeter on the scale, and the normal has been established by repeated tests at the laboratory.

The function of perception of light and motion is among the first developed of all ocular percepts. The quick and accurate perception of motion is very valuable to an aviator. As has been stated before, Nungesser, the famous French ace, said: "You need eyes all around your head and after a time, you get them."

With the subject placed fifteen inches from the test screen,



the threshold perception of motion covers a field about five degrees larger than that for the detection of form, but our tests at the laboratory show that the *field for the accurate* perception of motion direction is usually about three degrees less in all quadrants than the field for form, as *usually* taken.

Stereoscopic vision does not, according to our experience, suffer any very serious impairment in oxygen deprivation.

The test for the simple visual reaction time has not appeared to us to be of much practical value. In this test, the one possible reaction to the one possible stimulus becomes practically an automatic reflex. However, the Reeves visual

discrimination reaction time test is of much ophtholmo-psychological value. In this test there are four possible correct reactions to five possible stimuli. This act involves the higher mental processes and cannot become a simple reflex. In the laboratory, the average reaction time of this test is one half second, while the time given by many observers for the simple visual reaction is one nineteenth of a second.

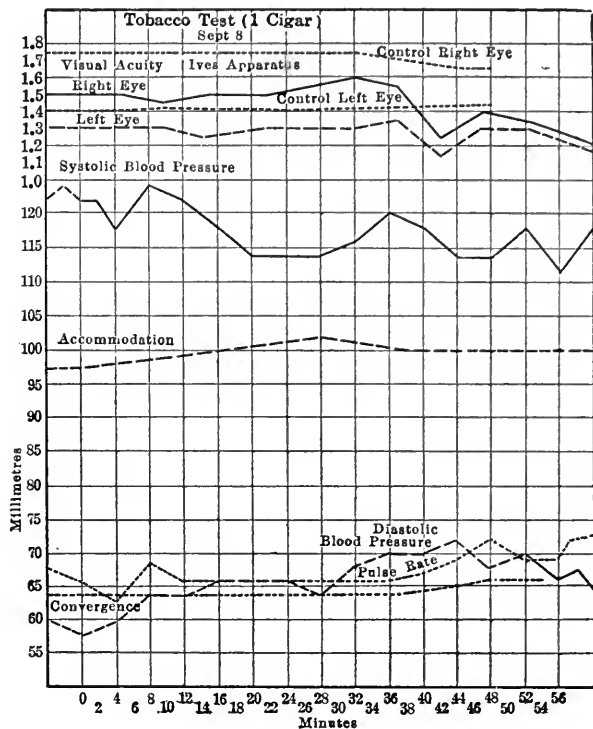
After all, the serious ocular disturbances produced in the peculiar environment of the airman take place in the intrinsic and extrinsic muscular apparatus. There is in many cases a falling off in the power of accommodation, convergence, and, naturally, in the field of binocular fixation. To illustrate this point, several diagrams are shown.

With the failure of accommodation, it is impossible to read the figures upon the ever-increasing number of instruments in the fuselage. The pilot has to shift his gaze quickly from the great expanse around him to the cockpit. Therefore his accommodation must be not only accurate but free from abnormal lag, otherwise the penalty is a broken machine, with the injury or death of the pilot. So, too, the failure in convergence means double, confused, and a danger-breeding vision.

While it does not bear directly upon the problems of aviation, as a bi-problem the intraocular tension has been tested under conditions of much reduced barometric and oxygen tension. In these tests no correlation has been found between these conditions, nor between the intraocular tension and the varying blood pressures occurring in a rarefied atmosphere. But more closely related to the problems of aviation are the effects of nicotine (or allied substances) upon the ocular functions. With reference to these effects upon the eyes and blood pressures of non-smokers, sixteen subjects have been tested in the laboratory, the smoking of one cigar being the time limit of the test. In twelve cases, there was loss in visual acuity; in one there was gain; while in three there was no change. In about one half of the cases there was a falling off in the motor apparatus of the eye. In regard to the interesting subject of blood pressure, it is sufficient to say, without going into details, that about 69% of the cases showed a rise in both systolic and diastolic blood pressure.

After months of serious thought and work on aviation problems, we have asked ourselves:

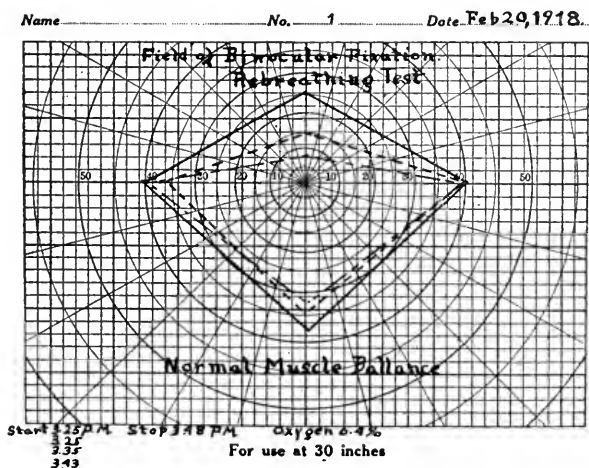
After all, what conclusions can one draw from this experience with an organ so wonderful in its fine adjustment that it can be compared, as Reeves has said, to a marvelous balance that can "with equal accuracy weigh a hair or a ton of coal"?



While the eye is only one of the peripheral sense organs concerned in the function of equilibrium, it is second to none in importance. We have found that its proper functioning is affected by oxygen want apart from the other trying conditions of flying, and we are more and more impressed with the vast importance of the initial selection of candidates who possess good eyes and well-balanced muscles, who have good adduction, and who are practically free from hyperphoria.



Equally important is the proper classification of the cadet, that he may be assigned to the type of work for which these laboratory tests have shown that he is best fitted. A cadet with low retinal sensitivity could not be a night bomber. A candidate who fails to read his instruments correctly and sees double at 10,000 feet could not be a pursuit or combat pilot. Once classified, the pilot should not be lost sight of.



The unceasing care of the flyer is the culminating duty of the flight surgeon.

A slack wire on a control is not as dangerous as a poor convergence. After the strain of service a tendency to a weak muscle becomes a manifest error. A weak convergence becomes a dynamic divergent squint.

By the aid of laboratory tests and by the constant, watchful care of the flight surgeon and of the physical director, many cases of incipient staleness can be detected and prevented, much economic waste can be prevented for the Government, while valuable young lives will be saved.

## THE NEED OF AN OPHTHALMOLOGIC CLINIC IN PENAL INSTITUTIONS. WITH SPECIAL REFER- ENCE TO ONE ESTABLISHED AT SING SING PRISON.

BY DR. CONRAD BERENS, JR., CAPTAIN, M.R.C., NEW YORK CITY.

**A**N experiment in sight-saving work was undertaken by the New York State Commission for the Blind, in April, 1916, through the establishment of an eye clinic in Sing Sing prison. This clinic was the outcome of two requests to the Commission from inmates. One man, who was blind, asked that some employment be found for him on his discharge, which would occur in a few weeks. The other, with a serious eye condition, asked the Commission to assist in securing a pardon in order that appropriate treatment might be instituted in time to prevent blindness.

Inquiry made in the interest of these two men disclosed the fact that a score of inmates of Sing Sing had serious eye affections for which the appropriation was insufficient to provide other treatment than that offered by the prison physician, and the services of an inmate jeweler who had been taught optometry.

The clinic was opened in April, 1916, and in less than a year became one of the most effective services which the Commission rendered the State in saving, not only eyesight for its citizens, but also dollars for its treasury, since blind citizens must often have supplementary assistance in industry and in maintenance.

Before the establishment of this clinic, an inmate optometrist was doing the refraction, which was assumed to cover the necessity for glasses and eye treatment in Sing Sing prison. From the cases under observation, it would appear that both

eyes were examined at the same time; therefore, if the vision of one eye was normal, both eyes were considered normal. In high myopia, the method was to increase the strength of the minus lenses as long as the patient said the letters looked "darker." Furthermore, numerous patients suffering from serious conditions, such as optic atrophy, cerebrospinal lues, detached retina, etc., received lenses but no local or general treatment. A study of the records shows that unless the sight of a patient was improved by lenses, he was thought not to need them. All of these conditions were to be expected, since the inmate who had been taught to refract knew nothing of general medicine, and could not legally use a cycloplegic.

This inmate optometrist was, however, a very careful and conscientious man, and with instruction in the technique of refraction gave valuable assistance in the development of the clinic. This important part of the clinical service was seriously threatened by the withdrawal on parole of this assistant; the situation was saved, however, as funds for his salary were obtained. This man has proved indispensable in developing many phases of the clinic's work. His value is reflected in the changed attitude of the patients, who have gradually come to a genuine coöperative spirit in recognizing the importance of the anamnesis concerning their physical condition and the relation of other physical ailments to eye conditions, as well as the importance of prompt and faithful treatment in preventing defective vision and possibly blindness.

At present the work is done in a large clinic room 25 x 15 feet, one corner of which has been curtained off as a dark room. The operations are performed in the general operating room. The clinic, dark room and operating room are as well equipped as many in the smaller hospitals, and, except for the difficulty in training men to observe the rules of asepsis, everything runs smoothly.

At the outset, it was obvious that the clinic should have proper histories, if good work were to be accomplished. Accordingly a card was devised which not only seems to fill most of the requirements of the work in hand, but is also revealing facts of more general interest. For instance: that a majority of these inmates have had less than seven years' schooling, that quite a considerable number are having their first educa-

tional classes within the prison. Many in each of these groups have had, since early childhood, myopic or other eye conditions which may have contributed to early delinquency and have been the means of substituting street trades for employment in such occupation as would furnish incentive for wholesome mental development.

In this phase of the service, another inmate has become indispensable, and by his services makes the clinic as efficient as many in the larger hospitals. This man is an expert stenographer, with more than the average layman's intelligence for medical facts. He is in constant attendance when the history is taken, at treatments and during operations, making accurate and detailed medical histories possible.

We have received the most cordial coöperation from all the officials at the prison and from the Department at Albany. Eye conditions are now considered in referring inmates for employment. For instance: a myopic patient is not sent to the knitting shop; and optic atrophy, iritis, and similar conditions exempt the inmate from employment which might be injurious to his eyes. Diagnosed eye conditions or suspicious symptoms of the same exempt inmates from draft to other prisons.

#### STATISTICAL REPORT

<i>Classification of Diagnoses</i>		<i>Classification of Operations</i>	
Marg. blepharitis.....	17	Pterygium, transplantation.....	1
Tumor of orbit (sarcoma).....	1	Pterygium, excision of.....	5
Ptosis, congenital.....	2	Trachoma, scarification, and exp.	1
Cellulitis of lid.....	1	Enucleations.....	13
Chalazion.....	18	Glaucoma, Elliot's trephine.....	2
Foreign body, lid.....	2	Senile cataract, preliminary iri-	
Atrophic globe.....	11	dectomy.....	1
Anophthalmos.....	9	Secluded pupil, iridectomy.....	2
Dacryocystitis.....	9	Glaucoma, iridectomy.....	4
Chronic follicular conjunctivitis	70	Traumatic cataract, needling...	2
Acute foll. conjunctivitis.....	10	Traumatic cataract, linear ex-	
Catarrhal conjunctivitis.....	5	traction.....	2
Foreign body, conjunctiva.....	2	Contracted cul-de-sac, plastic	
Keratitis.....	2	op.....	2
Pterygium.....	6	Dacryocystitis, excision of sac..	1
Old trachoma.....	14	Excision of papilloma of lid.....	1
Acute trachoma.....	1	Resections.....	7
Trachoma with pannus.....	1	Tenotomies.....	6

*Classification of Diagnoses*

Corneal ulcer.....	3
Anterior staphyloma.....	3
Abrasion of cornea.....	6
Macula corneæ.....	29
Burns of cornea.....	1
F. B. cornea.....	5
Iritis.....	11
Iridocyclitis.....	4
Traumatic cataract.....	6
Senile cataract.....	2
Dislocated lens.....	1
Chronic glaucoma.....	6
Retinitis.....	3
Choroiditis.....	3
Uveitis.....	3
Conv. strabismus.....	15
Div. strabismus.....	16
Alt. strabismus.....	10
Paralysis of superior rectus.....	3
Optic atrophy.....	13
Detached retina.....	3

Refraction

Hyper. astigmatism.....	65
Comp. hyp. astigmatism.....	18
Mixed astigmatism.....	28
Comp. myopic astigmatism.....	22
Myopic astigmatism.....	10
Hyperopia.....	130
Myopia.....	59
Presbyopia.....	40

*Classification of Operations*

Chalazion, scarification, & exp..	10
Ptosis, removal of redundant tissue.....	6

Of the 13 optic atrophy cases, 13 give a specific history, and 6 show dental infection in addition.

Of the 11 iritis cases, 8 give a specific history. Focal infection seemed to be the sole cause in two patients, and focal infection may have been a contributing cause in three others.

Uveitis: 2 show a specific history, with dental infection.

Total patients registered to date.....484  
Average weekly attendance at clinic..... 28  
Visits to clinic by patients.....2088  
Men sent from other prisons for eye treatment..... 10

TYPICAL HISTORIES

CASE 1. W. W., 28 yrs. Colored. Corrected vision: O. D.  $\frac{20}{30}$  O. S. fingers at 1 ft. Referred to ophthalmological department, June 5, 1916, complaining of failing vision in the left eye. A diagnosis of uveitis was made. He denied specific history and the Wassermann was four plus. He was referred to the dental department where several roots were extracted to permit alveolar abscesses to drain.

January 29, 1917. Eye quiet. Has received specific and dental treatment. Corrected vision: O. D.  $\frac{20}{30}$ ; O. S.  $\frac{20}{40}$ .

CASE 2. I. S., 29 yrs. White. Vision: O. D. no L. P.; O. S.  $\frac{20}{40}$ . Referred to ophthalmological department

December 18, 1916, complaining of pain in the right eye, blurred vision, photophobia, and lachrimation in the left. Injury to right eye twelve years ago. January 1, 1917, enucleation, O. D. January 16, 1917, O. D. wound healed. O. S.: eye quiet, no photophobia. February 27, 1917, given artificial eye. March 19, 1917, transferred to Great Meadows prison. June 20, 1917, sent another artificial eye, as the first was broken.

From a survey of the diagnoses, it will be seen that a large proportion of the patients referred are suffering from eye affections which are known to result disastrously if neglected or carelessly treated. It is a distinct advantage that patients giving a specific history and a positive Wassermann or such symptoms as make the diagnosis sure have not the freedom of the outside patient to refuse treatment, or to defeat successful treatment by migrating from one clinic to another. Syphilis is usually diagnosed at the time of the preliminary examination since the present system has been in force. Inmates committed prior to the establishment of routine physical examination are referred at the first warning symptom and treatment instituted. The benefit received by these patients is so generally recognized in the prison population that results are becoming much more satisfactory on this account.

Inmates, on commitment, are referred to the clinic as a routine measure. At the first visit the history is taken, the eyes thoroughly examined, and a careful preliminary refraction is made. We find upon examination myopic conditions which have been neglected since childhood, old trachoma capable of improvement by treatment, injured eyes needing enucleation to prevent sympathetic ophthalmia, beginning optic atrophy, and many other conditions requiring treatment. Refraction has been found to be the only treatment necessary in 282 cases. In a majority of the other cases, refraction has been included in the treatment; as in cataract, strabismus, and marginal blepharitis.

Of the patients referred, 35% give a specific history, and in a series of Wassermann tests taken as a routine on one hundred inmates, 31% were positive. In the table of diagnoses we have shown some of the eye conditions which prevailed in this group of syphilitics. One patient had five distinct attacks

of iritis in a period of nine months. That these two groups of inmates alone fully justify this service on the part of a Commission which would prevent blindness is seen from the case notes given from typical history cards.

In the prison population of fifteen hundred men, we have an average attendance of twenty-four on clinic day.

There are many men in the prison with normal vision in one eye, but not even light perception in the other, owing to a previous injury. They have never thought it necessary to have their eyes examined, since "they have one good eye." We have been able to emphasize the importance of enucleating the injured eye in order to preserve sight in the good one.

The other prisons in the State have sent ten men to Sing Sing for eye treatment—one for enucleation, on account of sympathetic ophthalmia.

To emphasize the importance of the plan for physical examination of prisoners on admittance, it may be interesting to mention some of the cases which came under observation at the eye clinic previous to establishing these examinations.

One of the most pitiful that I recall was of an inmate who entered the prison with a progressive myopia. He was given glasses which were, in all probability, too strong for him and in the absence of treatment detached retina had developed in both eyes so that he only counted fingers at two feet. At the time I saw him first, his iris showed no signs of inflammation; later there developed an acute iritis, and unfortunately he was not brought to me again until the iris was so bound down that mydriatics were of no avail.

Another, a colored man, was brought to my attention, who had been suffering for several days with an acute iritis. He was only receiving argyrol and ice. Atropin and hot bathing immediately relieved the situation.

The help we have received from the prisoners has been mentioned, but the work could not have been carried out if it had not been for the labor of Doctors H. R. Skeel, W. W. Weeks, and W. B. Weidler, who have freely given one afternoon a week, and of Miss Farley and Miss Clendinning, the Commission's nurses, who have been at Sing Sing regularly twice a week. Their devotion has kept this valuable work alive.

All who have assisted at Sing Sing say that it means more to them than any other work they are doing, for they feel that it is not right to put a man in prison and then waive all responsibility for him. We should keep each man physically fit and thus enable him to do some useful work. For this he might well be paid an ordinary wage, with an increase for greater production. In this way a man could pay for his keep in prison, and if he has a family he could help to support it. At present, each inmate receives one and one half cents a day. The saddest thing about sending a man to prison is the usual story about the family, left at home.

While the man is in prison, it has been our endeavor to help fit him for the work he hopes to take up when he is released. Operations are performed upon the eyelids and ocular muscles in the hope of improving the man's appearance and several operations have been performed to make a socket for a glass eye. By this means it is made easier for the prisoner to obtain a position after his release, and more important still, he is more likely to hold it.

A follow-up system is in force, so that men who need further treatment after release will be properly cared for.

From the foregoing it is evident that there should be an ophthalmologic clinic in every penal institution in the United States and everyone should do all in his power to help his brother on the inside, for he is worthy of it and needs us.



## THE RESULTS FROM 112 WASSERMANN BLOOD-TESTS IN THE MISSOURI SCHOOL FOR THE BLIND.

By DR. J. W. CHARLES AND DR. HARVEY D. LAMB, St. Louis.

QUESTIONS are frequently asked consultants in schools for the blind which can only be answered by reference to statistics of certain classes of cases which are more apt to be grouped in such institutions than in the private practices of ophthalmologists—*e.g.*: "What percentage of cases have been caused by syphilis?" "How great a rôle has heredity played in the production of blindness?" "What kind of cataracts yield the best results from needlings?" and even "Do you expect much result from optical iridectomy?" etc.

The class of cases terminating in such a school, coming from lowly families and often from ignorant physicians, makes manifest the difficulties of obtaining complete or accurate histories in some instances, *e.g.*, we have two children (sisters) from a county poor-farm who have, the one a coloboma and the other an aniridia. These children's antecedents cannot be traced at all.

In the semester of 1916 to 1917, 79 out of 106 pupils of the Missouri School for the Blind gave their consent for a Wassermann. Since the institution belongs to the educational system of the State, it seemed impossible to require the test of those who were unwilling. During this semester, however, we admitted no pupils without sending them to Dr. Jeans of the St. Louis Children's Hospital who enlisted the aid of the laboratory of Washington University in our 'cause, and we now have the results of 112 Wassermanns from among 137 pupils:

*Ophthalmia Neonatorum.* There were thirty-five cases of

which nineteen were born in the country or small towns while sixteen came from cities of more than thirty thousand. Of these, three rural and two urban cases refused the test.

Of the thirty remaining cases only one was positive, a child of underworld parentage.

Thanks to our present law there have been very few cases to be admitted in the last three years.

*Corneal Ulceration.* Of seven cases of simple corneal ulceration (necessarily indefinite) one refused the test, the other six were negative: three were from the country and two were urban.

The only case of *Parenchymatous Keratitis* was strongly positive.

*Trachoma.* We were surprised to find that only five pupils had been made blind by trachoma. Two refused the Wassermann and three were negative. All were rural.

*Malformations.* Of the twenty-one cases, sixteen were from the country, five urban. Five declined the test. Of the remaining sixteen two were without doubt syphilitic but had been treated and gave a negative reaction.

*Simple Trauma.* These four cases were all rural and all negative.

*Sympathetic Ophthalmia.* Four of the ten cases declined the Wassermann. Seven were from the country and three were urban.

The six submitting were all negative.

*Cataract.* Seven of the cataract cases were urban, eight were from the country. Three of the fifteen pupils refused. All of the twelve remaining were negative.

There were three pupils from one family of hereditary cataract and two from another.

*Progressive Uveitis.* It has been so difficult, sometimes impossible, to obtain a history of many of our cases that it has been out of the question to make a differential diagnosis of the various forms of uveitis which have terminated in blindness, and the work of obtaining a thorough investigation of the causes has been hampered in many ways.

Six of the seven cases were from the country and the Wassermann was negative. The one urban patient had undoubted stigmata of hereditary syphilis yet gave a negative blood and

spinal, but the patient had been treated with mercury by the stomach.

*Retinitis Pigmentosa.* Of the five cases diagnosticated as retinitis pigmentosa, all were from the country, two declined the Wassermann and the remaining three were negative.

*Retino-Choroiditis.* There were five cases, four rural. All were positive.

*Optic Atrophy.* Of the seventeen cases, nine were from the city and eight from the country. Three refused the test. Nine were negative, five were positive.

*Miscellaneous.* The one case of pemphigus gave a negative Wassermann. One case of *Retinal Detachment* at nine years with two blind brothers and a near-sighted sister gave a negative. One "congenital amaurosis" with keratoconus and ulcers declined. One "prenatal corneal scarring" with blue sclera gave a negative Wassermann. One man 44 years old with detachment of retina (with choroidal exudates, very high blood-pressure, albumin and hyaline casts) was reported positive a year ago; is now negative.

We thus have had from 112 Wassermanns, 96 negatives, and 11 positives besides five cases of undoubted syphilis

<i>Disease.</i>	<i>Total Cases.</i>	<i>Declined Test.</i>	<i>Neg.</i>	<i>Pos.</i>
Ophthalmia Neonatorum	35	5	29	1
Corneal Ulceration	7	1	6	0
Parenchymatous Keratitis	1			1
Trachoma	5	2	3	0
Malformations	21	5	14	2
Simple Trauma	4		4	
Sympathetic Ophthalmia	10	4	6	
Cataract	15	3	12	
Progressive Uveitis	7		6	1
Retinitis Pigmentosa	5	2	3	
Retino-Choroiditis	5			5
Optic Atrophy	17	3	9	5
Pemphigus	1		1	
Retinal Detachment	1		1	
"Congenital Amaurosis with Keratoconus and Ulcers"	1		1	
"Prenatal Corneal Scarring"	1		1	
Albuminuric Exudative Detachment of the Retina	1			1
	137	25	96	16

which had already received treatment and were negative, *i.e.*, sixteen cases of syphilis in 112 pupils. Remembering that the consensus of opinion of syphilographers is that 10% to 15% of the population have syphilis, and that at least 90% of hereditary syphilitic children give a blood-positive of which probably less than one half are made negative by treatment, we feel that our results have been accurate; and we believe that some interesting statistics along other lines could be compiled if those in charge of schools for the blind would co-operate by publishing as far as possible all cases of interest.

## REPORT OF A CASE OF EMBOLISM OF THE CENTRAL RETINAL ARTERY IN CHOREA.

By DR. ARNOLD KNAPP, NEW YORK.

R. K., aged 8, was seen July 6, 1917, stating that a month ago she had suffered from an attack of mumps, that condition being endemic in her neighborhood. The attack affected only one side of the neck, causing a swelling in the usual region without any febrile disturbance or malaise. One week later she developed typical choreic symptoms, consisting in a loss of control of the left hand and of the right foot, with twitching of the muscles of the face. On the following day the sight in the left eye was lost. About four days ago the skin of the lids of the left eye became black and blue for about twenty-five minutes and then this discoloration disappeared. She gives a history of repeated attacks of tonsillitis for several years and of mild joint pains for the past seven months.

The pupil of the left eye is dilated and does not react to light. No p. l. The optic nerve is white with a blurred temporal margin. The vessels are normal with the exception of the inferior temporal artery which is very thin. Pressure on the eyeball causes distinct arterial pulsation. The region at the posterior pole of the eye shows a haze of the retina which is particularly marked at the macula, this region appearing red (cherry spot) and presenting a number of bright spots and slight choroidal changes. The right eye is normal.

Dr. W. W. Herrick found, on physical examination, that the right tonsil was buried, the left apparently normal, large superficial lymph glands in the neck, with slight thyroid prominence. The heart is slightly enlarged to the right and in the pulmonary conus region. The first sound is short and abrupt and after exertion is prolonged into an extremely faint, short systolic blow. The second pulmonic sound is very loud. The rate is 100 and there is extreme sinus irregularity. From these signs he can not say definitely that there

is endocarditis. However, the enlargement to the right and in the conus region and the character of the first sound at the apex and of the second pulmonic are suggestive.

The case seems to be a comparatively straight one of an infectious parotitis followed by chorea minor and embolism of the central retinal artery. The examination of the heart revealed no anomaly and there have been no signs of articular rheumatism; the source of the embolism is an endocarditis of rheumatic origin, which is not sufficiently developed to give marked physical signs.

The association of chorea and embolism of the retinal artery has occurred in a number of instances, and Leber<sup>1</sup> contributes a chapter on this association. It seems clear that there is a closer relationship between embolism and chorea, for up to the present time Leber has collected nine cases exhibiting this association. The cases always occurred in youthful individuals between seven and twenty-six years of age. In three cases there was a pronounced cardiac lesion, and in two the cardiac symptoms were slight. In two cases articular rheumatism had preceded the eye disturbance. In some of the cases transient attacks of obscuration and blindness preceded the attack of permanent blindness and in one case published by Nettleship the attack of obscuration had also affected the other eye.

Leber, in the first edition of the *Handbuch*, was the first to draw attention to the probable association of embolism and chorea based upon the report of two cases. The additional cases which have since been observed substantiate the embolic theory of chorea. The close connection between chorea and acute articular rheumatism and endocarditis is founded on both clinical and pathological evidence. The pathological condition however is not clear. In some cases multiple emboli of the small cerebral vessels have been assumed and the corpus striatum and the thalamus opticus have been regarded as the areas affected. At the same time chorea may have other causes than cerebral emboli. The frequency of the combination of chorea with diseases which cause embolic disturbances is an

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<sup>1</sup> Leber, *Graefe-Saemisch-Hess Handbuch*, vol. vii., pt. 1, p. 271, 1916.

acknowledged fact. This is furthermore substantiated by the cases of sudden blindness in chorea which are presumably of embolic origin.

Oppenheim,<sup>1</sup> in discussing the various forms of chorea, speaks of the frequent association of chorea, articular rheumatism and endocarditis, and it is a striking clinical fact that chorea frequently follows acute articular rheumatism in which cases presumably the endocarditis is an intermediary. Occasionally the endocarditis is not observed until after the onset of the chorea, and the articular rheumatism also develops late. Articular rheumatism, however, has caused chorea without any evidences of endocarditis.

Some authors regard chorea as always of infectious origin, and a number of bacteria have been found on culture in these cases. Sachs<sup>2</sup> describes several cases of septic chorea with the presence of staphylococci and streptococci; finally, chorea has been occasionally found to follow certain infectious diseases, like scarlet fever, measles, and influenza.

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<sup>1</sup> Oppenheim, *Lehrbuch*, p. 1706, 6th edit., 1914.

<sup>2</sup> Sachs, *Medical Record*, 1908.

## HISTOLOGICAL FINDINGS IN EYEBALLS LOST THROUGH COMPLICATIONS FOLLOWING SCLERO-CORNEAL TREPHINING.<sup>1</sup>

BY DR. W. GORDON M. BYERS, MONTREAL.

(With two illustrations on Text-Plate XVI.)

THE following cases are of interest as showing the pathological changes in complications that apparently must inevitably occur at times after sclero-corneal trephining; and are instructive in that they bring out certain points in regard to the technique of Elliot's operation.

CASE I.—A woman, aged 31. Failure of sight in the left eye noted for several months; no other subjective symptoms. Pupil, oval in the horizontal meridian, fixed. Practically complete cupping of the optic disk, with marked pallor. V. = hand movements; a field (practically circular) of about 5° remains at the fixation point. T. = 34 Hgmm. O. D., fundus normal; F. of V. normal; V. = 90° + 0.75 + 0.25 %. T. = 15 Hgmm.

A sclerocorneal trephining was performed on March 2, 1915, with a complete iridectomy, easily achieved; no complications. The woman returned only on December 12, 1915, pregnancy and the birth of a child having made it impossible for her to come to the city at an earlier date. The eye had become suddenly painful shortly after leaving the hospital in March.

*Examination:* Eye unbearably painful and markedly congested; bleb collapsed, the broken conjunctival covering being mixed with prolapsed uveal tissue; no a. c. In the area of the coloboma upwards, occupying about a third of the space, is a small, tongue-shaped patch of corneal infiltration with its base resting on the limbus. Enucleation.

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<sup>1</sup> Read at the meeting of the American Ophthalmological Society, New London, July, 1918.



ILLUSTRATING DR. BYERS'S ARTICLE ON "HISTOLOGICAL FINDINGS  
IN EYEBALLS LOST THROUGH COMPLICATIONS FOLLOWING  
SCLEROCORNEAL TREPHINING."



FIG. 1.—Section through trephine aperture showing changes which are, in an exaggerated way, those that are characteristic of healing corneal wounds, with a marked response to injury or infection on the part of the uveal tract. The dislocated lens lies against the posterior opening of the wound, and its capsule is ruptured.



FIG. 2.—An ectatic scar covers dome-like the site of the trephine opening. It is lined for the most part by atrophied ciliary body, and its concavity is occupied by vitreous prolapse. The zonular fibers are ruptured, and the lens is displaced.



Microscopically one sees (Fig. 1) an open, apparently filtering wound. There are marked thickening, oedematous imbibition, and round-cell infiltration of the episcleral tissue. Toward the scleral side, the ruptured and thickened conjunctiva overhangs a very loose fibrillar reticulum. On the corneal side, large masses of granulation tissue are seen extending inwards, and especially downwards, from the thickened and bifurcated epithelial layer.

Behind, masses of pigmented cells are streaming forward into the wound around the promontory of the protruding sclera. On the corneal side, an enormous number of proliferated endothelial cells, with granulation tissue, are seen extending along Descemet's membrane in the immediate vicinity of the wound, and extending forward along the cut end of the cornea to plug the aperture.

The posterior opening of the wound is filled by the dislocated lens, save for a narrow channel on either side between the surface of this structure and the sclera and cornea respectively. The lens capsule is ruptured, and large numbers of reparative cells (epithelial as well as endothelial) are seen filling the gap, and infiltrating cleavages between the lens fibers.

Elsewhere one sees a well-marked plastic inflammation of the whole of the uveal tract with the usual changes. The cornea is markedly crinkled on its posterior surface towards its periphery.

The changes in this case are, in an exaggerated way, those that are characteristic of healing corneal wounds, with a marked response to injury or infection on the part of the uveal tract. In contrast to the second case, the dislocation of the lens and the tearing of its capsule in this specimen are attributable to the sudden reduction of intraocular tension following collapse of the bleb.

CASE 2.—A remarkably alert and well-preserved lady of 83. Gradual failure of vision for a few years, attributed to dense nuclear opacities of the lenses. These made a clear view of the fundi impossible. O. D., V. = fingers at 3 ft.; small field of vision only about the fixation point. T. = 40 Hgmm. O. S., V. = fingers at 20 ft.; F. of V. complete. T. = 23 Hgmm.

Sclero-corneal trephining, November 14, 1915; no complications, but slight difficulty in performing a partial iridectomy. The patient was perfectly comfortable until the morning of the eighth day, when three short, separate periods, in which flashes of light were seen, ushered in com-

plete loss of sight. Following this the eye became more and more irritable and painful, with greatly heightened tension, necessitating enucleation, January 22, 1916.

One sees in this case an ectatic scar covering domelike the site of the trephine opening. In all sections except the one here depicted, it is of almost uniform thickness, and composed of newly-formed fibrous connective tissue (still largely cellular) derived in part from the sclera, the cornea, and the uveal tract respectively. It is lined for the most part by the atrophied ciliary body; and its concavity is almost entirely occupied by a vitreous prolapse.

In the section illustrated a narrow, patent channel, immediately behind the cut surface of the cornea, leads from the posterior chamber to a dilated space directly beneath the conjunctiva. The epithelial covering of the latter is greatly attenuated, but the rupture seen occurred during the enucleation of the globe. In this specimen iris tissue is visible along the posterior surface of the cornea; but other slides show that a portion of the iris was actually removed, notwithstanding that elsewhere it is seen greatly atrophied and intimately adherent to the posterior surface of the cornea for a considerable part of its outer continuity.

The lens is displaced downwards; the zonular fibers are ruptured; and the vitreous prolapse here also blocks the filtering channel.

The point of interest in this case is the vitreous prolapse, and a brief study of the specimen shows clearly what must have been the course of events leading to its occurrence. The trephining in the first instance was too peripheral, though the operation was performed after considerable experience with the procedure, and it seemed quite satisfactory at the time. During the first few days the uveal tract was sufficiently strong to support the intraocular structures; but it gradually stretched under pressure, and finally gave way on the eighth day, with consequent rupture of the zonular fibers and hyaloid membrane, dislocation of the lens, and renewed tension following vitreous prolapse. At the time of the occurrence of the flashes of light the feeling was, that they were probably attributable to retinal hemorrhages; but there are no extravasations of blood to be seen in any part of the specimen.

The lesson here is obvious. Especially in these advanced cases in which accidents of this sort are prone to occur, Colonel Elliot's advice in regard to placing the trephine opening in corneal tissue should be followed to the fullest extent. Only in this way can one guard as far as possible against the occurrence of the changes described; while on the other hand the procedure offers the best chance of securing unadherent iris for excision.

I am indebted to Dr. Gross for the preparation of the illustrations.

## SPASM OF ACCOMMODATION<sup>1</sup>

BY DR. JAMES W. WHITE, NEW YORK.

THE accompanying case of spasm of accommodation is reported because of the infrequency of the condition and of some unusual elements in this case.

The etiological factors of spasm of accommodation are more or less agreed upon by various observers. Koenigshoefer classifies them under the following heads. (*a*) Those of central origin, functional neuroses, general intoxications, etc. (*b*) Reflex spasms, neuralgia of the fifth nerve, muscle anomalies, affections of the lids and conjunctiva, sinusitis, etc. (*c*) Traumatic spasm (rarely direct trauma to the ciliary body but usually reflex). (*d*) Occupation spasms. The classification of Mauthner is essentially the same as that of Koenigshoefer.

The condition is most frequently met in hysteria and is more frequently seen in myopia than in hypermetropia or emmetropia. The cases reported as coming on after sudden fright (Venneman) and accompanying dysmenorrhœa (Mooren) are of group (*a*) in the classification. Donders saw no cases of spasm of accommodation in 2000 consecutive cases of myopia. Koenigshoefer saw four cases in 34,000 cases of myopia and reported eight cases in all irrespective of refractive error. Graefe reported a case in 1854 without refractive error that required - 13 D lens for distance, and Liebreich reported the case of a young woman twenty-one years old who accepted - 13 D for distance but who could see clearly only at a distance of six inches. Under atropine her refraction was + 0.25 Sp.

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<sup>1</sup> Presented at the meeting of the Section on Ophthalmology, New York Academy of Medicine, February, 1918.

Spasm of accommodation is occasionally produced by the local instillation of eserine.

Spasm of accommodation like paralysis of accommodation is usually of cortical or nuclear origin. If cortical it will affect both eyes simultaneously. If nuclear it may affect one eye or both according as one or both nuclei were involved. Spasm of accommodation due to trauma may be unilateral if the trauma is such as to cause direct or reflex irritation of one ciliary muscle.

Several reported cases of spasm of accommodation are, as pointed out by Koenigshoefer, not true spasm of accommodation but are an unusually powerful accommodation within normal limits. The term "spasm of accommodation" is often improperly applied to cases that respond slowly to a cycloplegic or to those who on a post-cycloplegic test accept a much weaker lens than when under the cycloplegic. Yet it seems difficult to distinguish these from spasm. A man who persistently accepts an overcorrecting concave lens is certainly exhibiting a spasm of accommodation.

**CASE REPORT.**—Miss E. L., age 27. Nine years ago began to have poor distant vision and diplopia. She was examined by an ophthalmologist who said she required  $-3.50$  D lens. He refused to prescribe this but used atropine instead. Thirteen months later he found  $-9$  D necessary but again advised against the use of glasses. Soon after this she was seen by another ophthalmologist who prescribed  $-6.50$  D lenses. Two and a half years later this was raised to  $-8$  D and sixteen months later to  $-9.00$  when  $10^\Delta$  base out R. and L. was combined with this. This was worn constantly until June, 1917. At this time her vision was R. and L.  $\frac{20}{60}$  without glasses corrected to  $\frac{20}{10}$  R. and L. with  $-9$  D. With this she read J. #1 at 10 inches and without glasses J. #1 at 6 inches. There was an alternating esotropia of  $46^\Delta$  for distance and  $50^\Delta$  for near and an homonomous diplopia of the same amount. Associated movements showed an insufficiency of both externi and of associated movements upward. The pupils were moderately dilated but reacted normally to light and convergence. Atropine was prescribed for refraction. Three days later under atropine the following was found: V. R. and L.  $\frac{20}{60}$  + without glasses. Skiascopy R. +  $0.25$  +  $0.25^\circ$   $90^\circ$ , L. +  $0.25$  Sp. She now had an esophoria of  $1^\Delta$  for distance and  $8^\Delta$  for near. There was no diplopia at any

distance. External recti and associated movements were normal. No glass was prescribed but atropine was continued for one week when it had to be discontinued on account of systemic poisoning. While under the cycloplegic + 2.50 lenses are necessary for near vision. Frequent attempts have been made since to use atropine or scopolamine but neither can be used for more than a week or two. The condition has remained essentially the same for the past year. The amount of esophoria or esotropia varies from time to time depending on the degree of cycloplegia and returns to the original amount when the cycloplegic is discontinued.

Complete neurological examinations have been made at the Neurological Institute and at the neurological department of Vanderbilt Clinic without determining the cause.

Treatment thus far has been the continued use of cycloplegics with diverging exercises. When not under cycloplegic a reduction in the strength of the glasses and discontinuing the use of prisms. Thus far the condition has remained stationary.

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REPORT OF SEVERAL PATHOLOGICAL PUPILLARY  
CASES, WITH ESPECIAL REFERENCE TO WHAT  
CONSTITUTES A TRUE ARGYLL-ROBERTSON  
PUPIL.

BY DR. JOHN DUNN, RICHMOND, VA.

THE objects in the report of the following cases, interesting as they are in themselves, are (1) to further accentuate the fact that the *primary pupillary response to light is an autonomic reflex* and as such belongs to the vegetative system, and, *further, is mediated through the ciliary ganglion*; (2) to call attention to what constitutes the true Argyll-Robertson phenomenon, and (3) to explain how and why the array of symptoms constituting the uncomplicated Argyll-Robertson syndrome vary from case to case and in the same case at different stages of its history.

In the May number of the ARCHIVES OF OPHTHALMOLOGY appear two papers, one on "The Eye in Nervous Diseases" by Dr. W. A. Holden; the other on "The Light Pupillary Reflex, Its Path and Abolition," by Dr. Anton Lutz. Both papers overlook the possibility of there being such a thing as a pupillary response to light which is not aroused by impressions which have traveled from the retina along the optic nerve to the photomotoricus nucleus of the third nerve and thence outwards.

The ciliary ganglion finds no place in the diagram of the visual pathway as given by Dr. W. A. Holden (p. 233). Dr. Lutz devotes several paragraphs to the importance of the ciliary ganglion but apparently sees in it only a transmitter, so far as constriction of the pupil is concerned, of impulses which have come from the third nerve centers. Nowhere have I been able to find answers to the simplest questions about the

*raison d'être* of the ciliary ganglion, and until these answers have been correctly given the existing confusion in regard to the primary pupillary reflex must continue. Why do the motor fibers to the pupil and ciliary muscle pass through a ganglionic mass in the orbit (the ciliary ganglion) and not go direct, as do the motor fibers, to the external ocular muscles? For what purpose do the sensory and sympathetic fibers enter this same ganglion? Why do the long ciliary nerves not find a ganglionic connection within the orbit necessary?

Heretofore all efforts to explain the primary pupillary response to light have started with the effect of light on the retina and following the retinal fibers centralwards have tried to make connections which would explain the primary, consensual, and accommodative reflexes. The result has been, as Bumke confesses, that "the exact determination of the pupillary reflex tract is not only the next but the most important thing to be determined." Let us leave out of consideration for the moment the optic nerve path. Let us confine our attention to the sensory and motor terminal fibers from and to the ciliary ganglion. The short ciliary nerves consisting of sensory, motor, and sympathetic fibers emerge from the ciliary ganglion and are distributed to the cilio-iritic region. Sensations picked up by the sensory elements of the short ciliary nerves are carried backwards to the ciliary ganglion. Why to the ganglion? This is the crux of the whole question. The ganglion is not a purposeless mass of nervous tissue. It has a definite function. What is it? Why is it that the motor fibers, the sensory fibers, and the sympathetic fibers that enter it do not pass with their forms unaltered through the ganglion? Why do all three sets of fibers make connections with each other within the ganglion? There can be but one answer—and that is that it is to make possible the existence of primary reflexes; in the case of the ciliary ganglion, these reflexes are manifested in movements of the pupil and ciliary body and in changes in the activities of the ciliary gland. Primary reflexes are brought about wherever there exist extra-cerebral ganglia. The jerk of the foot when the patellar is struck is a reflex response, and to explain it we do not say the nervous pathway is sensory nerves to the spinal cord, along the sensory spinal nerves to the cerebral cortex where communi-

cation is made with the motor cells and thence through the internal capsule to the spinal cord and along the motor tract outwards to the leg muscles. We are satisfied to say that the knee jerk is mediated through the appropriate spinal ganglia. Admission of this in no way lessens our understanding of the willed and coördinated movements of the foot and leg. This reflex belongs to the domain of the voluntary muscles. The primary pupillary response belongs to the domain of the autonomic system. Or, look at the question another way, the sensory elements of the short ciliary nerves convey sensory impressions born in the iridociliary region to the ciliary ganglion. These impressions must serve a physiological purpose within the ganglion, otherwise the ganglion would be unnecessary. The finer ganglionic anatomy is such as to render it certain that these sensory impressions are transmitted to both the ciliary and iritic motor fibers within the ganglia. Then what happens? Motor fibers do not transmit impulses centralwards. *The impulses then must be transmitted peripheralwards and can have but one effect, contraction of the sphincter pupillæ and of the ciliary muscle.* The sensory impulses from the iridic and ciliary region, which on reaching the ciliary ganglion are therein communicated to the sympathetic element, are transmitted also peripheralwards and result in a response as the part of both the activities of the ciliary glands and of the dilatator pupillæ. If these statements are not so, what is the purpose of the ciliary ganglion? I can conceive of no reason why the motor fibers to the iris and ciliary muscle should be interrupted by a peripheral ganglion other than that thereby is rendered possible the production of primary reflexes, *i.e.* those reflexes that are produced without recourse to the higher nervous centers. The long and short ciliary nerves are distributed to the ciliary and iritic regions. No sensory nerves have been demonstrated as going from the retina. With an intact iris and ciliary body, if the retina be entirely atrophied, it is impossible to produce the primary light reflex. This proves beyond question that an active retina is a *sine qua non* for the production of the primary light reflex. The retina has distributed to it no sensory nerve terminals from the ciliary ganglia. We might, at first thought, then exclude the possibility of the ciliary ganglion mediating the primary light reflex as claimed above. When,

however, light falls on the retinal ganglion cells, great activity is aroused in the adjacent pigment cell layer. This layer is continuous to and through the ciliary and iritic regions, and it is through disturbance of this pigment layer that the sensory nerves of this latter region are stimulated for the originating of the primary light reflex. The motor cells to the ciliary muscle also pass through the ciliary ganglion. This has also for its purpose the making possible of a primary ciliary muscle reflex. This is also possibly secondary to the impact of light in the retina, just as is the primary pupillary reflex, and has for its object a reflex contraction of the ciliary muscle occurring at the same time with that of the contraction of the pupil. It is more likely, however, that the primary ciliary reflex is secondary to the iritic movements of the primary light reflex. These movements create sensory impressions which are transmitted to the short ciliary nerves, thence outwards along the ciliary motor fibers from the ciliary ganglion to the ciliary muscle. *This is also an autonomic reflex and is entirely distinct from the accommodation act.* As the primary pupillary reflex is probably protective in its origin, so too is this primary ciliary reflex. *This primary ciliary reflex is not possible when there is complete atrophy of the retinal cells, and yet under the same conditions both contraction of the ciliary muscle and its consensual pupillary response are at times possible (vid. Case No. 1).* Why do the long ciliary nerves not pass through the ciliary ganglion? Because their impulses do not take part in the production of a primary reflex. When the ciliary muscle begins to contract, and thus is put in motion the crystalline lens system, a set of sensations is aroused different from those aroused by the impact of light on the retina. It is these sensations that are taken note of by the long ciliary nerve terminals and carried centralwards to inform the brain of the degree of contraction of the ciliary muscle, information which assists in regulation of the accommodative action of the crystalline lens *and in accompanying changes in the size of the pupils.* This is the origin and explanation of the movement of the pupil in accommodation.

In considering the anatomy of the ciliary ganglion in the light of the claim made that for the production of the A.-R. pupil the syphilitic organism selects for its action the sensori-motor and the sensori-sympathetic connections within the

ganglion, it may justly be asked, how is this possible? I am inclined to think the explanation is to be sought in the method of termination of the ultimate arterial supply to the sensory nerve terminals. It is probable that we have supplied to each element of the ganglia end-arteries, just as occur in the retina.

It must be borne in mind that what we have written here bears directly only upon the primary pupillary response to light. The paths for the consensual light reflex and the accommodative pupillary response are different and much more elaborate, requiring the aid of the third nerve nuclei and higher centers for their accomplishment and regulation.

The first case to be here reported is one of the *Pseudo-Argyll-Robertson Pupil*.

*That in a given case the pupils do not respond to light either directly or consensually and do respond in convergence does not necessarily make the case one of the Argyll-Robertson pupil.* The true Argyll-Robertson pupil, free from complications and before degenerative changes affecting either the retina or the motor fibers in the ciliary ganglia set in, gives the above syndrome *plus the fact that the pupils are contracted*. Elsewhere I have tried to show that the lesion causing this picture can be situated nowhere else save in the ciliary ganglia and can be caused only by microorganisms or their products which shut off therein the sensori-motor and sensori-sympathetic connections. So far syphilis is the only proven cause. Where a lesion of this character exists the sensory nerve impulses from the ciliary and iritic regions to the ciliary ganglia do not arouse corresponding responses in the motor fibers to the ciliary muscle and to the iris, the result being absence of the primary or autonomic reflexes of both the iris and ciliary muscle. Further, no action on the part of the sympathetic within the ganglion is possible and the only impulses reaching the iris are along the motor fibers passing directly through the ganglion. A contracted pupil is the necessary result.

CASE I.—James F., negro, aged 50. Examination showed both eyes to be totally blind; both pupils were fully dilated and gave no trace of response to light either directly or consensually. The negro being blind could not converge his eyeballs. When told, however, to try his best to look at the end of his nose, both eyes would turn downward and slightly

inwards. As the result of this effort, both pupils distinctly contracted; dilating when the eyes returned to their usual position. This case is interesting as showing (1) that the crystalline lens system may remain active for a considerable time after an eye has become completely blind following complete retinal atrophy; (2) where the retina is completely atrophied the primary pupillary light reflex is abolished, as is (3) the consensual light reflex, but not (4) necessarily the accommodative consensual pupillary reflex. In this case the patellar reflexes were present and while the neuroretinitis, which caused the total blindness, may have been syphilitic in origin, I could find no other confirmatory symptoms. The convergent accommodative pupil response, when the experiment mentioned above was made, was well marked. In my experience the retention of the accommodative pupillary response in totally blind eyes cannot be demonstrated, save in rare instances of which this case is one. The reasons are not difficult to understand. The retention in this case makes the case worthy of report.

This case shows one type of the pseudo-Argyll-Robertson pupil. W. A. Holden (*loc. cit.*) says that "non-luetic tumors in the region of the third ventricle by pressure have produced typical Argyll-Robertson pupils." I have never had the opportunity of examining the pupils in a case of a tumor thus situated, and it is impossible for me to understand how such a tumor can produce a typical Argyll-Robertson pupil. Every fact of the history of the growth of brain tumors and every fact of the history of the Argyll-Robertson pupil pleads against such a possibility. The typical A.-R. picture may show for years contracted pupils, normal vision, and no associated paralysis of any external ocular or other muscle. A "non-luetic tumor in the region of the third ventricle" has no such chapter in its ocular history. Such tumors may produce bilateral pupillary disturbances but at no period of their history do these disturbances correspond in all details to the typical Argyll-Robertson pupils. It is impossible that such should be the case. Bilateral disturbance of the "association fibers" between the "external geniculate body, the anterior quadrigeminal body, and the optic thalamus" and "the nuclei of the third nerves causes reflex iridoplegia (A.-R. pupil)" (W. A. Holden, *loc. cit.*). If the light impulses which are carried centralwards by the optic fibers are prevented from reaching

the third nerve nuclei, why should the pupils be contracted (at times almost to a pin point), as they are in a case of uncomplicated A.-R. pupil? The pupils do not contract when both optic nerves have been cut. They do not contract when both optic tracts have been cut. Why should they contract when these same stimuli sent into the central optic centers have been "disturbed" on their way to the third nerve nuclei? The fact is they do not. If the lesions are of such a character as to cut off the transmission of the optic nerve impulses to the third nerve nuclei, then dilatation of the pupils will result. That the belief that a lesion situated in the region of the third nerve nuclei can produce a typical, true A.-R. pupil is so general in ophthalmological neurology is due to misunderstanding of what constitutes a true A.-R. pupil. There are other reasons why "bilateral syphilitic lesions in the region of the external geniculate bodies, the anterior quadrigeminal bodies, and the optic thalamus" cannot produce uncomplicated typical Argyll-Robertson pupils.

CASE 2.—Illustrating how injury to the third nerve may cause paresis of the constrictor pupillæ as its only demonstrable result.

John B., aged 9, three weeks prior to consultation was pulled backwards from his chair, striking the back of his head against the floor. For a short while he was stunned and finally vomited. Nothing else was observed except that when his father, who is a physician, examined his eyes he found that the right pupil was dilated. This condition, persisting for three weeks, the boy was brought to me. Examination of the right eye showed a normal fundus; normal accommodation; no demonstrable external muscular unbalance; field of vision normal; normal vision. The right pupil was semi-dilated, but responded to light directly, consensually and in accommodation. The left eye was normal. The boy whose intelligence was of a high order, stated that he felt no inconvenience as the result of the blow, *except that he seemed to have some difficulty in judging distances*. In this case, I think the right third nerve was stretched against the edge of the dura where it enters the outer wall of the cavernous sinus, when the brain was thrown forward. The stretching of the nerve resulted in injury to the pupillary fibers sufficient to result in a partial dilatation of the pupil but insufficient to produce a complete paralysis or to prevent the pupillary responses. The "difficulty in

judging distances" is to be attributed to a similar injury to the other third nerve fibers, an injury insufficient to produce diplopia but sufficient to prevent the quick, exact and equal responses of the external muscles of the two eyes. This unilateral isolated paresis is, I believe, not uncommon as the result of injuries to third nerve trunk from injury similar to the one described above, but also, from pressure upon the nerve due to growths and syphilitic deposits at the base of the skull. In especial should this latter be borne in mind when we are endeavoring to determine the nature of the pupillary inequality in any given case of the Argyll-Robertson pupil. This has been commented upon elsewhere and will be returned to later on in this paper. A similar paretic condition of the sphincter may be brought about by a blow on the eyeball.

CASE 3.—A case of unilateral dilatation of the pupil accompanied by reactions, *the reverse of the Argyll-Robertson Pupil*.

Mr. A. L., aged 25. Examination of the right eye revealed the following: paresis of the levator palpebræ, internal rectus, superior rectus, inferior obliquus and inferior rectus. The paresis being greater in superior rectus and inferior obliquus than in the other muscles; less in the internal rectus than in the superior rectus and inferior obliquus. There was complete paralysis of the accommodation, and had we examined the eye without the aid of a strong magnifying lens we would have said that there was complete paralysis of the constrictor pupillæ. The pupil, however, was only semi-dilated. The fundus was normal. The vision was normal. Field of vision was normal. The left eye was normal. The patient had "suffered with" his "head" ever since he could remember. At the age of eighteen he had consulted an oculist, who, as far as I could learn from the patient had found present this incomplete paralysis of the third nerve. At all events, for at least seven years the patient had been conscious of double vision, which is instantly made worse by any effort to look at an object above an angle of about 45 degrees. The cause of his consultation was to seek relief from the periodic severe headaches which seemed to center in and about the right eye. These headaches were worse the greater the interval between them. As to a cause of the paresis, there was only an indefinite history of a severe blow across the upper part of his nose when he was two years of age. On casual examination one would have said all three of the right sided pupillary reflexes were absent. When, however, the pupil was examined under a magnifying glass, there were to be seen distinct responses to light, directly and consensually. These re-



sponses were weak, but unmistakable, the entire circumference of the pupil responding. On the other hand no response could be made out in accommodation; in other words, when a finger held before the nose was gradually approached toward the eye the left pupil would contract, but, although the paretic right internal rectus would turn the eyeball partly inwards, there was no visible response on the part of the right pupil. The interest of this case for us here lies in the pupil. The dilatation is not complete. This tells of a lesion posterior to the ciliary ganglion. There is present the primary light reflex; this shows that the retina, the pigment layer, and the sensory nerves from the ciliary region to the ciliary ganglia are still intact. It shows that the communication within the ganglion between these sensory nerves and the motor efferent fibers to the pupil are still intact. That the pupil contracted shows that responses from the third nerve center still reach the ciliary ganglion; that the response is a feeble one indicates that the amount of the impulse from the third nerve reaching the ganglion along the pupillary fibers is subnormal; *i.e.* that there is partial "paralysis" of these fibers. That the consensual reflex should exist, but be also feeble is also readily understood; but *there is no pupillary response in accommodation.* This is due to complete paralysis of the accommodation posterior to the ciliary ganglion.

CASE 4.—*A case of a type often reported as one of Argyll-Robertson Pupil.*

In reading cases reported, especially by the neurologists, one not infrequently finds among the symptoms the following "Argyll-Robertson Pupil Optic Atrophy," without further ocular details. The case to be reported here is instructive. James B., aged 34. Cause of consultation inability to read. General examination shows loss of patellar reflexes. Both pupils are semi-dilated; about equal in size; both are slightly oval in shape; *apparently* there is no response either directly or consensually to the light; patient can converge well, *apparently* also there is abolition of the accommodative reflex. Why I say *apparently* will in a moment be understood. Vision O. D. Light perception. O. S. 18/50  $\geq$ . The field, O. D., is more or less concentrically reduced to a very small area, somewhere near the macula. The exact limits of this area cannot be defined, because of the poor vision. (The test was made in a dark room with a candle.) The left eye is blind for almost all of the upper half of the field and part of the lower. Both optic nerves show a high degree of atrophy, with contraction and unevenness of outline of both arteries and veins. Patient is unable to read even with glasses. When the pupils are

examined in the usual way no movement can be seen. The case, however, is not one of the Argyll-Robertson Pupil. If, under a strong magnifying glass, the pupil be carefully studied for a while there can be made out a faint hippus and from time to time feeble responses to light both primarily and consensually and in accommodation. These responses are, however, not constantly present and care and patience are necessary to demonstrate them. Their presence, however, is beyond question. The lesion in this case is not in the ciliary ganglia, even though the patellar reflexes are absent. We are dealing with the late stages of a case of syphilitic optic atrophy, in which although most of the retinal ganglia have disappeared some few still remain, and the blood supply to the remaining few is very poor, as is shown by the condition of the veins and arteries. With the disappearance of the retinal ganglia there is a disappearance of the activity of their appropriate pigment cells. Thus it has come about that the sensory stimuli to the ciliary ganglia are very feeble. Both sets of stimuli, however, still exist, but they are both too feeble to produce more than the extremely faint responses. The field of vision asks several questions about the extent of the lesion whose answers are out of place here.

CASE 5.—*A case of uncomplicated Argyll-Robertson Pupil.*

Mr. L., aged 54, initial lesion twenty odd years ago. Absent patellar reflexes. Vision, both eyes, normal. *No disturbance of the secondary act of accommodation.* Both pupils are as contracted as though he were suffering from opium poisoning. Entire absence of primary and consensual light reflexes. Retention of accommodative convergence reflex. (To see this, however, the eyes have to be viewed under a magnifying lens.) Fundi normal. Media normal. Fields of vision normal. Above is written no disturbance of secondary act of accommodation. In all cases of true Argyll-Robertson pupil, the primary reflex response of the ciliary muscle is abolished just as is primary pupillary reflex. Just as the primary response to light is not a part of the act of conscious vision but is protective in its character, so the primary ciliary reflex is not a part of the accommodative act but is also protective in its character, inasmuch as has to do with regulating the outflow of the aqueous as disturbed by the primary pupillary reflex. This primary ciliary muscle reflex is abolished because the sensory fibers of the short ciliary nerves from the ciliary muscle to the ciliary ganglia cannot deliver their impulses because of the specific disturbance in the sensori-motor ganglionic connections as explained above when writing of the primary pupillary reflex. In this case the accommoda-

tive action of the ciliary muscle is not interfered with. The sensory link for the regulation of this action is furnished by the long ciliary nerves whose entire tract is uninterfered with.

CASE 6.—*Argyll-Robertson Pupil complicated with unilateral disturbance of the third nerve fibers.*

Mr. A., aged 38, initial lesion at age of 20. Absent patellar reflexes; severe gastric crises; epileptiform attacks of unconsciousness, etc. O. D. Paralysis of all the muscles, external and internal, including the levator, supplied by the third nerve. Pupil semi-dilated. Loss of all pupillary reflexes. Loss of all accommodative reflexes. External rectus and superior obliquus intact. Vision normal. Field of vision normal. Fundus normal. O. S. Pupil contracted. Entire absence of primary and consensual pupillary reflexes to light. Retention of convergence accommodative reflex. Accommodation normal. Vision normal. Field normal.

This is a case of paralysis of the right third nerve, occurring along with the Argyll-Robertson pupil. The dilatation of the right pupil is caused by the paralysis of the third nerve. That there is not dilatation *ad maximum* shows that we are dealing with a disturbance of the third nerve posterior to the ciliary ganglion. It is not nuclear. The ciliary muscle being completely paralyzed and the act of convergence rendered impossible for the right eye all of the accommodative reflexes are abolished. The left eye shows a typical A.-R. pupil. This case is reported because it illustrates one of the causes of the unequal pupils of the A.-R. type. The normal A.-R. pupil picture can be further disturbed by lesions of the nuclei of origin of the third nerve; by lesions affecting the external rectus, or the superior obliquus, unilaterally or bilaterally, and from various sources. These things should be borne in mind when trying to interpret the deviations from the normal A.-R. pupil.

There is still another chapter in the history of the A.-R. pupil and it produces a well-known picture, although its interpretation has hitherto been at fault. The claim is made by the author that the true A.-R. pupil is the result of a selective lesion within the ciliary ganglia, a lesion limited in its first stages to the sensory terminals of the sensori-motor and sensorisymphathetic ganglion cells. If this be so, what happens when the syphilitic agents transgress their primary limits and

involves the motor elements? In the first place a dilatation of the hitherto contracted pupils takes place, equal or unequal for the two eyes as the process within the ganglia is equally or more advanced in one ganglion than the other. What happens when the syphilitic process has destroyed the motor nervous elements of the ciliary ganglia? Dilatation of both pupils; abolition of the primary pupillary and ciliary reflexes; abolition of the consensual light reflexes; abolition of the accommodative pupil reflexes; degeneration of the short ciliary nerves to the ciliary region and later the degeneration of the pigment layers of the retina, degeneration of the retinal elements and an increasing amblyopia and finally total blindness. This condition is not the same as shown in Case 4.

So far as I know, no careful comparative study of the size of the pupil and its responses in the various stages and types of glaucoma has ever been made. There is one picture of the number, however, that is of especial interest here, inasmuch as it lends its weight to our explanation of the primary pupillary response to light. This picture presents itself in certain cases of glaucoma which slowly progress to blindness before the eye becomes the seat of either great pain or of an acute inflammatory attack. It is characteristic of these cases that the pupil is smaller than normal, of equal size with that of the other eye, and that there is relatively slight lessening of the depth of the anterior chamber. Let us take, for example, the case of Mr. L., aged 70. Right eye, vision normal. Left eye, tension  $+ 1\frac{1}{2}$ . Totally blind. Anterior chamber not shallower than that of many old people without glaucoma. Pupil about  $1\frac{1}{4}mm$  in diameter; is absolutely irresponsive to light; responds actively both consensually and in convergence. At first thought one would say there is nothing unusual about this; that the eye does not respond to light because no light impulses pass along the optic nerve to awaken a response, while the convergence and consensual reflexes keep the two pupils of equal size. Such an answer, however, is probably not the whole truth. In the earlier history of this case there almost certainly existed such a condition as is found in the left eye of Mrs. P., aged 56. Right eye normal. Pupil  $1\frac{1}{4}mm$ . Left eye, tension  $+ 1\frac{1}{2}$ , pupil  $1\frac{1}{4}mm$ . Total abolition of response to light, with retention of both consensual and con-

vergence reflexes. Vision, however, is distinct perception of light for a portion of the lower part of the field. Here then is an eye which perceives light and in which both the consensual and convergence reflexes are retained and yet the eye shows complete absence of the primary pupillary response to light impulses. Here we have an eye whose optic nerve is capable of transmitting centralwards impulses which awaken consciousness in the sight centers and yet when these impulses reach the sight centers they arouse no corresponding response in a pupil which is capable of normally responding both consensually and in convergence. If this case be correctly observed—and I used much care in its observation—then it seems to prove that the primary pupillary response to light is a reflex which neither originates in nor is controlled by either the mid-brain or the cortical ocular centers. What is the explanation of the abolition here of the primary light reflex? In the cases both of Mr. L. and Mrs. P. the glaucomatous process had lasted over a number of years, during which time the increasing pressure had destroyed not only the retinal ganglion cells but the activity of the retinal pigment cells. The activity of these cells is a *sine qua non* for the production of the primary pupillary response to light—as we have tried to show elsewhere. In considering the cortical brain centers of sight and their connections we must bear in mind that they were not added to the primitive brain for the purpose of responding to light impulses. This was something of which the primitive brain was capable. They were added for the purpose of making possible the interpretation of form and color and of bringing their combinations under the domain of the memory. A more detailed discussion of the disappearance of the primary response of the pupil to light before the disappearance of light perception by the eye is reserved for a later paper.

## CAVERNOUS SINUS THROMBOSIS. WITH NOTES OF FIVE CASES.<sup>1</sup>

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**T**HROMBOSIS of the cavernous sinus is the rarest of large sinus thromboses, less than three hundred cases having been published since its recognition by Herman Knapp (1) in 1868. MacEwan (2), whose description of the condition is one of the best, reports but four cases which he has himself seen. Occurring so seldom in the practice of any one man, early decisive recognition may easily fail. Since, however, the only possibility of lessening the extremely high mortality of the disease lies in prompt surgical interference, such early decisive diagnosis becomes necessary. A brief review of the salient facts known about cavernous sinus thrombosis is therefore presented, based upon the literature, which for so rare a condition is quite voluminous, upon five cases from the practice of the writer which are here first reported, and upon ten cases published since Langworthy's study in 1915.

### NOTES OF FIVE NEW CASES.

CASE I.—S. S., a Polish male of 25, was admitted to the Bridgeport Hospital, April 16, 1915. Previous history negative. Present history: Seven days ago swelling appeared on the bridge of the nose, extending to both eyelids, especially the left; no obvious infection; no history of trauma. Slight nasal discharge for past three days. Complains of chills and headache only. Temperature on admission at 4 P.M. 102.8°. Physical examination shows a well nourished man with marked exophthalmos of the right eye and complete immobility of the globe. Pupil dilated and insensible

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<sup>1</sup> Thesis, American Ophthalmological Society, 1918.

to light. Some swelling of the lids and chemosis. Left eye normal. Pain and tenderness over the entire face and slight stiffness of the neck. Physical examination otherwise negative.

*April 17.* Temp. A.M. 101°; P.M. 105°; restless, complaining of violent headache. Rhinologist reports "Nothing in nasal passages." Slight swelling of left upper lid was noted at 5 P.M.

*April 18.* Temp. A.M. 103.4°; P.M. 105°. Marked exophthalmos and fixation of left globe all day. Symptoms in right eye receding. Spinal fluid reported by Dr. Peters: "Pressure slightly increased, turbid, 500-600 cells per *cu. mm.*, mostly polymorphs, globulin + +; Fehling present, no organisms." Blood culture shows staph. aureus bacteremia. The patient was semiconscious in the morning, unconscious in the afternoon. 8 P.M. temp. 106°. At 8.55 he died.

*April 19.* Autopsy of head only permitted. Report by pathologist, Dr. Peters. "There is considerable chemosis of tarsal conjunctiva, especially of left eye, but no exophthalmos. On removal of brain there is found a thin purulent exudate over base. In the cavernous sinus of the right side is found a somewhat purulent bloody substance; no firmly attached thrombus. Ethmoid cells and frontal sinuses normal; mastoids normal. Smears from base of brain and from sinus show numerous Gram-positive cocci, mostly in pairs."

CASE 2.—Mrs. H. G., an American woman of 35, was seen in consultation with Drs. Cogswell and Roller at 1 A.M. August 15, 1915. Previous history: negative except for chronic right ethmoid infection. Present history: chills and high fever for several days, with severe headache and meningeal symptoms; swelling of right eye thirty-six hours ago and of left eye beginning eight hours ago. Delirious for the past six hours. Phys. exam.: Patient unconscious but very restless. Marked exophthalmos of both eyes with swelling of the lids, intense chemosis of the conjunctiva, and complete immobility of both globes. Pupils dilated and insensible to light. Fundi show considerable congestion and dilatation of veins. Temp. 106°. One hour later she died. No autopsy permitted.

CASE 3.—W. H. L., an American male of 35, was seen in consultation with Dr. D. C. Patterson on October 11, 1915, because of a conjunctivitis accompanying a facial erysipelas which apparently started from a fissure in the right nostril. The conjunctivitis cleared and, owing to absence from the city, the man was not seen again. Upon returning, his physician reported that the erysipelas ran a typical course and was very much improved, when, October 27th, he be-

came much worse; the right eye became proptosed, the pupil dilated, and there was marked chemosis and lid swelling. Three days later exophthalmos of the left eye appeared and there were symptoms of meningitis. October 31st, he died.

CASE 4.—Miss A. W., an American woman of 60, was seen in consultation with Dr. Cogswell, October 4, 1916. Previous history: negative except for an abscess at the root of the left eyetooth which had been under treatment for the past four weeks. Present history: two days ago there was a slight swelling of the left upper lid which slowly increased; aside from this she seemed well until this morning, when her sister thought she was a little flighty in conversation at breakfast, and insisted upon putting her to bed and sending for the doctor. Dr. Cogswell came at noon and found her semiconscious, restless, with a high temperature and with some exophthalmos of both eyes. At 3 P.M., when I first saw her, there were marked exophthalmos of both eyes, chemosis, and complete fixation of both globes, pupils dilated and fixed, retinal veins greatly dilated. She had a high temperature, was unconscious and could not be roused. At 9 P.M. she died. No autopsy permitted.

CASE 5.—T. M., a German of 38, seen in consultation with Dr. Roller, December 4, 1916. Previous history not obtained. Present history: sickness began with a boil on the left side of the nose seven days ago. This was incised and dressed dry. Two days ago he had a chill and high fever and meningeal symptoms appeared. This morning exophthalmos of the left eye was noticed and he became irrational. This afternoon the right eye also was involved. When I first saw him at 9 P.M. there were moderate exophthalmos of both eyes with complete loss of motility; dilated pupils; fundi normal except for congestion; some doughy swelling below and outside the left eye, apparently not connected with the boil on the nose, which was healing; patient unconscious and quiet with deep stertorous breathing. Four hours later he died. No autopsy.

NOTES OF TEN CASES REPORTED SINCE 1915, OR NOT MENTIONED IN PREVIOUS STUDIES.

Wacker's case (18) followed a furuncle inside the nose. Exophthalmos on the third day, meningitis the fifth, death on the seventh day. Autopsy showed staphylococcic thrombophlebitis of both cavernous sinuses and metastatic abscesses in the lungs.



Henning's case (19) was also from a facial infection, as was another case reported in the discussion.

Dayton (20) reports two cases; one from a purulent infection in the nose with death eleven days after eye symptoms appeared; the other in a girl of 15 who had typhoid fever and a probable lighting up of a chronic nasal sinus disease; death in seven days. No autopsies.

Payr (21) diagnosed a sterile thrombus in the cavernous sinus following an infective thrombophlebitis in the upper arm of the opposite side. There was no exophthalmos, but all the nerves in the sinus were involved.

Davis's case (22) followed tonsillitis, with death in ten days. Exophthalmos of one eye only. Spinal fluid normal. He has seen two other such cases after tonsillitis, one recovering with loss of sight, the other dying on the fifth day. Also a case with double exophthalmos following mastoid disease and operation, with death on the fifth day. In the discussion O'Malley reported a case three weeks after tonsil and adenoid removal; autopsy showed osteomyelitis of the sphenoid, probably the primary focus. St. Clair Thompson has seen three cases, two from the sphenoid and one from the ear. J. Horne had seen one sphenoidal case. F. Powell one case secondary to intracranial suppuration.

Sitzen (23) reports two cases; the first followed a boil on the nose with death ten days after the appearance of the boil, three days after exophthalmos involved the second eye. Autopsy showed thrombosis of the cavernous and superior petrosal sinuses and meningitis from staphylococcus infection. The second case followed a facial erysipelas starting in a nasal furuncle. There was exophthalmos of both eyes and pronounced meningeal symptoms. Autopsy showed cavernous sinus thrombosis and meningitis. Knoch reported a third case, also from a boil on the nose, with death on the third day. No autopsy. These three cases occurred in Java.

#### SURGICAL ANATOMY.

The cavernous sinus, the most important venous channel at the base of the skull, lies between layers of the dura beneath the temporo-sphenoidal lobe. It is roughly quadrilateral in shape, larger behind than in front, 2cm or more in length and

about 1cm in diameter. Evidences of its origin as a venous plexus in the dura remain in the interlacing bands of lining endothelium, which divide it into several irregular compartments. It extends along the side of the sella turcica, separated by a very thin bony wall from the sphenoidal sinus of the nose, from the sphenoidal fissure at the apex of the orbit where it receives the ophthalmic vein, to the apex of the petrous portion of the temporal bone where it opens into the petrosal sinuses, the superior to the lateral sinus, the inferior to the jugular vein. The cavernous sinuses of the two sides are connected by the circular sinus, two transverse vessels one in front of and one behind the pituitary body. On the internal wall of each sinus lie the internal carotid artery and the sixth nerve; on its outer wall from above downward the third, fourth, and the ophthalmic division of the fifth nerves. Each cavernous sinus has emissary veins through the foramina ovale, lacerum medium, and Vesalii to the pterygoid and pharyngeal plexuses, and a plexus of veins around the artery in the carotid canal to the internal jugular vein. It also receives veins from the frontal lobes of the brain and from the middle cerebral vein.

The cavernous sinus is thus in direct venous connection through the lateral sinus with the veins of the ear, through the pterygoid plexus with the veins of the mouth and throat, and through the ophthalmic vein with the veins of the orbit, of the nose, and of the face. The facial vein, on account of its exceptional patency and lack of valves, favors septic absorption and thrombus formation, and the extension of thrombi therefrom to the cavernous sinus is anatomically very easy, either via the angular and ophthalmic veins, or via its tributary from the pterygoid plexus. The orbital veins are also valveless. The inferior ophthalmic, which has a branch through the sphenomaxillary fissure to the pterygoid plexus, joins the superior ophthalmic, which communicates with the angular vein at the root of the nose and receives blood from the ethmoidal veins and the *venæ vorticosæ*, and empties into the cavernous sinus, together with the central vein of the retina.

#### ETIOLOGY.

According to MacEwan purulent otitis is the commonest cause of cavernous sinus thrombosis. Next to this are infec-

tive lesions of the orbit and face, *i.e.*, of the area drained by the ophthalmic vein and its branches. Of these, orbital cellulitis, boils and infected wounds on the face, and facial erysipelas are the most common. Other causes are purulent affections of the nose and accessory sinuses, especially the sphenoid and the ethmoids, pharyngeal and tonsillar infection, and abscesses at the roots of the teeth. Thrombosis may occur from trauma in the immediate vicinity of the cavernous sinus, as in head injuries. A non-infective thrombosis of the cavernous sinus has been described by Knapp.

The following table shows the original source of infection in 140 cases.

	<i>Ear</i>	<i>Orbit and Face</i>	<i>Mouth and Throat</i>	<i>Nose</i>	<i>Other Causes</i>
Dwight-Germain (4)	43	40	14	9	
Jackson (5)	13	6	2		4
Cases here reported		3	2	4	
Approx. %	40%	35%	13%	9%	3%

*The path of infection.* Infective meningitis from caries of the body of the sphenoid may extend by contiguity, according to MacEwen (2), directly to the cavernous sinus, as may intracranial suppurative processes. Ordinarily however, there is set up in the veins draining the infective source a thrombophlebitis, from which the thrombic process extends through venous channels to the cavernous sinus. Its usual routes are from the sigmoid through the petrosals from behind, through the ophthalmic veins in front, or from below by way of the pterygoid plexus.

The infecting organism may be any one of the pus-producing group, of which perhaps the staphylococci and streptococci are most frequently found.

#### SYMPTOMS.

The general symptoms are those of any infective sinus thrombosis, headache, chills, fever with a steep-peaked chart,

quick soft pulse, and euphoria. Occasionally there is vomiting. Leucocytosis is present; blood infection may or may not be found. Spinal fluid changes, if present, are those of a beginning basilar meningitis, which is not uncommon. Pulmonary complications from septic emboli in the lungs are frequent. The later symptoms are those of a rapid profound sepsis, delirium, then coma, preceding death.

The local symptoms are largely eye symptoms, and occur in two groups: I. Those due to intraorbital venous obstruction, and II. Paralyses of the nerves within the sinus, due to pressure and to phlebitis of the sinus wall.

Of the first group the most important as well as one of the earliest symptoms is exophthalmos. This according to most observers is rarely absent; Knapp (6) states that it occurs in 72% of the cases. It appears first in the eye upon the same side as the originating infection, rarely on the opposite side, and increases as the infection advances. The eyeball is pushed forward by the venous engorgement and œdema at the back of the orbit, which also cause swelling and œdema of the lids extending over onto the root of the nose, chemosis of the conjunctiva, and engorgement of the conjunctival veins. This may be well marked within a few hours. If the thrombic process involves the pterygoid plexus, there may be œdema of the pharynx and tonsil, but this is easily overlooked unless the latter was the source of the infection. There may also be œdema of the face from obstruction of the facial vein and of the tissues of the temple and about the ear. All these obstructive symptoms vary in amount in different cases, being particularly marked where there is ophthalmic phlebitis; they may entirely disappear with the development of the collateral circulation. | |

Shortly before death the same symptoms usually appear in the other eye, often with a recession of symptoms in the first eye. The involvement of the second eye indicates the extension of the thrombus through the circular sinus to the cavernous sinus of the other side, and makes the diagnosis practically certain. MacEwen (2) says that involvement of the second eye occurs in more than half the cases. One hundred and thirty-four autopsies collected by Dwight-Germain (4) showed

only thirty-one in which the thrombosis was limited to one cavernous sinus.

The second group of local symptoms comprises the various paralyses of the nerves within the cavernous sinus. The third nerve is most often implicated, ptosis being one of the most constant symptoms, though often not available for diagnosis on account of the great œdema of the upper lid. Strabismus varies, depending upon the priority of involvement of the third, fourth, and sixth nerves, and soon gives way to complete ophthalmoplegia.

The various paralyses may occur early and are best observed in the cases of slow development, particularly those in which the infection comes from behind. In the more acute cases and in those in which the infection comes via the ophthalmic vein, the displacement and fixation of the eyeball from intraorbital pressure often occurs before any distinct paralyses can be recognized. The pupil, small at first, is widely dilated, sluggish, then immovable. The cornea becomes hazy and anæsthetic, and may break down from exposure. Increased lacrimation, according to Weeks (7), is soon followed by comparative dryness of the conjunctiva. Neuralgic pain in the first division of the fifth nerve may occur, but is much less common than headache.

Vision fails or is lost according to the pressure on or stretching of the optic nerve. Its measurement however is usually impracticable on account of the general condition of the patient. In four cases which recovered, quoted by Jackson (5), only one had good vision, and in Johnson's (9) and Davis's (22) cases vision was lost.

The ophthalmoscopic symptoms are not ordinarily important and may be lacking, probably because of the free anastomosis of the ophthalmic veins with the facial and with the pterygoid plexus. If the fundus can be seen, fullness and tortuosity of the retinal veins is usually found, with perhaps slight blurring of the margin of the disk; neuroretinitis and retinal hemorrhages may occur. These are symptoms of obstruction of the ophthalmic and retinal veins rather than of cavernous sinus thrombosis. The course of the disease seems ordinarily too rapid to allow the development of a well marked optic neuritis.

## DIAGNOSIS.

Septic thrombosis of the cavernous sinus will be thought of whenever any exophthalmos shows also septic and cerebral symptoms, or when any case of probable cranial sinus thrombosis develops orbital symptoms. The combination of lid swelling, conjunctival chemosis, exophthalmos, and paralysis of a nerve within the sinus on the same side as an infective lesion anywhere about the head is quite characteristic, and the diagnosis is made certain by the similiar involvement of the other eye.

When the infection comes from behind or below, the process extends from the cavernous sinus to the orbit, and the general symptoms of sinus infection precede the local or orbital ones. Here the first appearance of the characteristic local symptoms makes the diagnosis easy, and an earlier diagnosis is possible than in the cases infected from the front. In the cases where the infection is by way of the ophthalmic vein, as for example where the sinus involvement originates from a previous orbital cellulitis, orbital symptoms precede the sinus infection. Here the involvement of the sinus is marked by the deepening of the symptoms of the cellulitis, and the addition of those of cavernous sinus infection. In these cases it is more difficult to be sure of the diagnosis until the second eye begins to be involved.

The precise order in which the symptoms appear may thus depend upon the source and path of the infection. Familiarity with the possibilities of infection in this region, and a clear mental picture of the pathology of sinus thrombosis, are of more service than statistics of symptoms in making an early diagnosis.

## DIFFERENTIAL DIAGNOSIS.

Septic cavernous sinus thrombosis is to be differentiated from other pathologic processes within the cavernous sinus. These, according to Langworthy (3) are: Injuries from gunshot wounds, from fractures at the base of the skull, and from operations on the Gasserian ganglion, the hypophysis, and the sphenoidal sinus; arterio-venous aneurism; malignant or congenital new growths involving the sinus; marasmic or aseptic thrombosis; and perhaps intermittent exophthalmos. In all

these the history, the rate of development, and the other symptoms should afford basis for diagnosis.

Infective sinus thrombosis is to be further differentiated from pathologic processes within the orbit. Of these orbital cellulitis and orbital thrombophlebitis limited to the orbit (the latter, according to Weeks (7), occurring practically only in erysipelas) offer the most difficulty. These have, however, no cerebral symptoms unless the process extends to become either sinus thrombosis, meningitis, or cerebral abscess.

Exophthalmos and other symptoms which might possibly cause confusion are also present in: 1. Tenonitis, which is a much milder condition, often suggesting rheumatic infection and associated with trauma or with infectious disease rather than with an infective focus. 2. Periostitis, with thickening and tenderness of the orbital periosteum and with less interference with eye movements. 3. Panophthalmitis, with the infection mainly inside the globe. 4. Dacryo-adenitis, a rare affection. 5. Infection of the nasal accessory sinuses adjacent to the orbit. 6. Exophthalmic goiter. 7. Tumors and cysts of the orbit, of the optic nerve, and of the lacrimal gland.

#### COURSE.

In infective cases the obstructive symptoms, exophthalmos, lid oedema, and chemosis, develop early and gradually, and ordinarily reach their maximum in two to five days, rarely ten or more, when they begin to subside. The development of the process in the second eye is a late symptom and is much more rapid, being often a matter of hours. The nerve symptoms come early and are soon masked by the obstructive symptoms. The sepsis grows more profound, and embolic processes, especially in the lungs, are frequent. Meningeal symptoms and those of increased intracranial pressure often develop. and death regularly follows from sepsis in six to forty-eight hours after the involvement of the second eye.

#### AUTOPSY FINDINGS.

Autopsy findings by various observers agree only in that the cavernous sinus contains a thrombus, often partially disin-

tegrated, or a purulent fluid; that usually both sinuses are involved; and that there is frequently a basilar leptomeningitis. Other findings depend upon the source, route, and nature of the infection.

#### PROGNOSIS.

The process is always fatal if the thrombus is infected and is not drained. If the symptoms arise simply from an obstructing sterile clot by extension from the sigmoid sinus, and this clot does not become infected because of early operative interference on the sigmoid, recovery may be possible. This has actually occurred in three cases quoted by Dwight-Germain (4), in one by Adair-Dighton (8), and in one by Johnson (9). Bircher (10) went through the petrous portion of the temporal bone and drained pus from the posterior end of the cavernous sinus, successfully. Langworthy (3) quotes three recoveries from drainage of the infecting source only; in these it seems fair to assume that the thrombus in the cavernous sinus was not infected.

In some such way must the few recoveries, where septic thrombosis of the cavernous sinus has been diagnosed, be explained. The total of recoveries in nearly three hundred cases<sup>1</sup> reported in the literature amount to twenty-three, about 7%. Eliminating those recoveries which may be explained as above, and considering the great number of observers, the fragmentary character of many of the reports, and the possibilities of inaccuracies and of errors in diagnosis, we are forced to the conclusion that thrombosis of the cavernous sinus is practically always fatal if the thrombus is infected and is not drained.

#### TREATMENT.

Except for drainage of the infective source, and drainage of the lateral sinus in ear cases, the treatment of cavernous sinus thrombosis is so far practically nil. The only hope of effectual treatment lies in the analogy of this condition to thrombosis of the lateral sinus, where early diagnosis and prompt, bold, and

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<sup>1</sup> The exact number of cases reported is uncertain, as collections by different authors contain a few of the same cases, and sufficient data is not always given to surely identify all of these.



thorough surgical intervention saves many lives. Theoretically, if the cavernous sinus were attacked exactly as the otologist attacks the lateral sinus, by free incision, removal of the clot or its septic part, and drainage, some cases should recover, especially those in which diagnosis and operation are made early. The difficulties in such a procedure are obvious. Lateral sinus infection is common and its operative technique has been well developed, while cavernous sinus thrombosis is too rare to allow any surgeon to see more than a few cases. By the time the diagnosis is usually made and the question of operation arises, the sepsis has progressed so far as to make the chances of recovery slight. The cavernous sinus, too, is surgically one of the least accessible structures in the body, and few operators care to explore it. The feasibility of its drainage has, however, been demonstrated in operations by Hartley (12), Dwight-Germain (4), Ballance (13, 27), Ballance and Hobhouse (13), and Voss (14), all of whom approached the sinus by the Hartley-Krause Gasserian ganglion route, through the side of the skull; by Mosher (15) and Adair-Dighton (24), going through the orbit; and by Bircher (10), in an ear case, going through the petrous portion of the temporal bone to the posterior end of the sinus. The last case was the only one to recover. This, however, is not a fair measure of the success of the operation. In some cases the operation came too late to relieve the sepsis; in some the sinus itself was missed. In Hartley's case a sarcoma was found in the sinus, and this was the cause of the patient's death two months later. In Dwight-Germain's case, age 40, incision into one sinus completely relieved the interference with the circulation in both sinuses. While drainage of one sinus in Adair-Dighton's case, age 6, did not drain the other, the original focus in the nose was overlooked.

Other routes to the sinus seem possible. Luc proposes an extension of his operation for exposure of the maxillary antrum. Beck has successfully operated hypophyseal tumors by way of the antrum, following Jansen's method. Langworthy (3), as a result of cadaver study, proposes a nasal route through the roof of the sphenoidal sinus, a route which Hirsch and others have successfully followed in operations upon the pituitary body in acromegaly.

Of the possible routes by which the cavernous sinus may be surgically reached, that from in front is in the field of the ophthalmologist, those from below in that of the rhinologist. The brain surgeon has developed entrance through the side of the skull, and one otologist has reached the sinus from behind. The weight of evidence so far favors the Gasserian ganglion route, though the route through the sphenoidal sinus may be more attractive to those who have gained experience in this region by operating upon the pituitary body. It may prove that the choice of routes will be that with which the operator is most familiar; or perhaps that by which the infection has entered. The observations of military surgeons in injuries of the cavernous sinus may throw light upon the question. Meanwhile we do not know how many of these theoretic considerations are practical.

Operative interference has so far few advocates and fewer practitioners, though its feasibility is admitted and it seems to be the only rational treatment when it has been better worked out. With many surgeons of experience the objections to operation outweigh its advantages, though this fact may be discounted by the natural conservatism of the profession toward all unproven measures. Opinion is not unanimous. Many agree with De Schweinitz (26), who thinks that "operation would seem to be proper," and with Frazier who is quoted by Pooley (25) as saying that "Direct attack on the cavernous sinus should be made in the hope of saving more lives."

Langworthy (3) makes the point that until we have a better understanding of the very early signs of the disease, we cannot expect to improve in either diagnosis or surgical treatment. This should be gained by a more general alertness and more careful observation in that class of infections from which thrombosis of the cavernous sinus may develop.

#### CONCLUSIONS.

A study of septic cavernous sinus thrombosis indicates:

- I. The hopelessness of any but surgical measures, first to the source of infection, second to the sinus itself.
- II. Septic cavernous sinus thrombosis is an operable

condition. The sinus should be opened and drained if a definite diagnosis can be made before the patient becomes too septic.

III. An early definite diagnosis can be made in at least some cases, if the original observer is familiar with the condition and is sufficiently alert. Discussion of the subject may therefore be of value, as many cases doubtless go unrecognized. More careful study of the earlier symptoms is needed, and the possibility of sinus thrombosis developing from apparently trivial infections about the face and head should be kept in mind.

IV. A larger number of cases operated upon is necessary before more definite conclusions can be drawn. And in view of the hopelessness of non-operative measures and the possibility of saving an occasional case by early and thorough drainage of the sinus, the operation should be undertaken whenever there is any reasonable possibility of success.

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## ACUTE STREPTOTHRIX INFECTION OF THE CONJUNCTIVA.<sup>1</sup>

BY DR. ARNOLD KNAPP and DR. JAMES G. DWYER, NEW YORK.

CLINICAL REPORT. Miss W. U., aged 30, while nursing a delirious patient who was suffering from uremia, cystitis, and pernicious anæmia, received some of the expectoration in her left eye. The patient had a very bad typhoid mouth. On the afternoon of the following day, March 30th, the eye became inflamed and itchy. On March 31st the inflammatory symptoms were aggravated and the discharge became purulent. In the afternoon the nose and throat became sore and the preauricular gland enlarged. The eye was treated with salt irrigations and argyrol without any improvement. When I saw the patient on April 2d, the lids of the left eye were red and swollen, extremely tender, particularly in the region of the retrotarsal folds. On everting the lids the conjunctiva presented a large number of follicles which were larger than those seen in trachoma, more separated, and some showed a yellow tinge. The whole left half of the face was tender; the auricular gland was enlarged and sensitive. The patient was then referred to Dr. Dwyer for a bacteriological examination. On April 3d the condition of the eye was worse and she was advised to enter the eye hospital. The nose caused a great deal of distress. On that afternoon there was not much secretion from the conjunctiva, and the follicles were about the same. On inspecting the pharyngeal wall and the regions posterior to the palatal arches, the same type of follicular inflammation was observed.

The treatment of the eye consisted in silver nitrate, boric acid irrigations, and iced aluminum acetate pads. There was a gradual improvement, particularly from April 7th. The follicles slowly disappeared from the conjunctiva and

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<sup>1</sup> Presented before the Section on Ophthalmology, New York Academy of Medicine, March, 1918.

the swelling of the preauricular gland decreased. The pharyngeal condition was also much improved. On April 10th the conjunctiva of the eyelids was still red and thickened, but the follicles had disappeared. The swelling in front of the ear had gone and the throat was much better. The patient, however, was extremely languid and still suffered from the after-effects of considerable poisoning.

On April 17th, Dr. Dwyer reported that the smears were all negative. The cultures from the eye, nose, and throat gave a growth of streptothrix. The culture from the eye was pure but those from the nose and throat were mixed with other organisms.

From the literature, the streptothrix is usually associated with chronic conditions of the eyes, and it is rare to find it as an acute infection. However the method of infection was so direct here that the throwing in of an organism of even low virulence in such a manner would easily give an acute condition. The analogous condition of sporothricosis, described by Schenck and others and which is more common, is usually an acute infection but the morphology of the organism is very dissimilar and can easily be differentiated.

#### SCIENTIFIC REPORT:

##### *I. Morphology.*

1. Direct examination shows rod-shaped organisms with typical true branches. Very occasionally the end of the branch shows a club-shaped end.

2. Subcultures morphologically show almost the same appearance with however the branches more clearly marked. True branching appears. The organism is Gram-positive and is easily stained by the ordinary watery dyes.

##### *II. Cultural.*

Growth very scant on meat infusion agar of .5% acidity. Growth much better on glucose meat infusion agar. Growth on gelatine the same as agar and gelatine is not liquefied. There is nothing characteristic on any other media, the whole appearance of the growth resembling that of other members of the higher bacteria.

##### *III. Animal pathogenicity.*

1. Local inoculations—rabbits.

(a) Rubbing into the skin was negative after two months.

(b) Rubbing into conjunctival sac of one eye was followed in ten days by an intense conjunctivitis, with "steaming" of the cornea and then an ulcerative keratitis which went on to loss of the eye.

(c) Inoculation into the anterior chamber of the other eye was followed by a low grade hypopyon, lasting for two weeks with a gradual extension as a cyclitis and loss of the eye. This process lasted over one month.

(d) Subcutaneous inoculation was followed by the formation of several subcutaneous abscesses of a very low-grade indurated character and which took three weeks to develop. These abscesses were of the "cold" variety, were apparently painless in character. Upon evacuation of these abscesses, a thin colorless fluid was obtained, which contained flakes floating here and there. These flakes morphologically showed the streptothrix, as described above. The animals did not die until three and one half months after inoculation and at this time the abscesses were still of the low indurated variety but the contained pus was very thick and creamy with very few organisms. At autopsy the animals (two) showed general emaciation and deposits in the spleen and in the lung in one of them.

Through the bronchoscope, a broth culture of the organisms was blown down into the left lung. This was followed by a pneumonia on that side in five days, an extension to the right and death in six days. At autopsy, the organisms were recovered from both lungs and the typical appearances of pneumonia were present.

## 2. Inoculations—guinea-pigs.

With the exception of the eye experiments, the same findings were apparent in four pigs.

# REPORT OF THE ANNUAL CONGRESS OF THE OPHTHALMOLOGICAL SOCIETY OF THE UNITED KINGDOM.

BY MR. HENRY DICKINSON, LONDON.

The annual Congress of this Society took place in London during the three days May 2d to 4th, under the presidency of Mr. E. TREACHER COLLINS, F.R.C.S., and, despite the war and the consequent transfer of a large proportion of the medical talent into the Army, may be voted a distinct success, both in regard to the attendances and the quality of the contributions submitted. Most of the formal discussions took place at the rooms of the Royal Society of Medicine, but, in addition, a clinical meeting was held at the National Hospital for the Paralyzed, in Queen Square, and a visit was paid to the Metropolitan Asylums Board Ophthalmia School, at Swanley in Kent, of which the President is Consultant, and an interesting discussion took place there on **contagious diseases of the conjunctiva**.

Advantage was also taken of the presence of so many ophthalmologists from the various parts of the Kingdom to hold a meeting for the purpose of forming a Council of Ophthalmologists to watch and take measures concerning the national well-being from this special standpoint. There was complete unanimity, and the speeches of leading members of the specialty favored a stiffening of the qualifications to practice the special branch. It was agreed that the Council shall consist of all the past and present presidents of the Ophthalmological Society of the United Kingdom and of the Royal Society of Medicine Section of Ophthalmology (these to be permanent members), four members, elected annually, from



the Councils of each of these bodies, and one representative of the Oxford Ophthalmological Congress.

THE PRESIDENT'S ADDRESS.

Mr. COLLINS, in opening the proceedings, said the Society was not a body which awoke into activity on three days in the year, and lay dormant during the remaining 362. Matters of ophthalmological interest requiring constant vigilance and care were constantly arising, and he instanced the question of visual standards for the Army, and the more energetic manufacture and supply of Captain Cruise's visor for military use. The War Office had recently created the post of Consulting Ophthalmic Surgeon to the Forces at Home, and he congratulated Colonel Herbert Parsons on being appointed to that important position. Numbers of men had, during the war, been transferred from countries in which contagious ophthalmia was perennially rife to others in which it was of only sporadic occurrence. Many men, free from any contagious eye disease had been sent into districts where, it was estimated, 90% of the population had trachoma, or had had it at some time. These men would, in due time, return and mix with their own uninfected kith and kin. He thought some concerted action should be taken to prevent a repetition of what took place—infection on a large scale—on the return of our troops from Egypt after the Napoleonic wars. He referred to the cohesion among ophthalmologists which had been brought about by affiliation of provincial societies and by the merging of various ophthalmological journals into one. There was still ground for dissatisfaction, he said, in regard to the visual tests for the performance of various duties in the public service, and for the carrying on of certain occupations in which defective eyesight might prove injurious. Blindness arising from the curable condition ophthalmia neonatorum was still far too frequent, and further efforts were required in the direction of caring for the eyesight of children during the unnatural visual strain of school life, especially in the provision of curricula for short-sighted pupils. He made a strong appeal for a definite training in ophthalmology before medical diplomats were allowed to practice the speciality. He concluded by

announcing that the 1919 Bowman Lecture would be delivered by Dr. Morax, of Paris, and that the Edward Nettleship Gold Medal and Prize had been awarded to Lieut.-Colonel Gordon Holmes, M.D., for his valuable work during the last three years on disturbances of vision in association with cerebral lesions.

### **Effects of Hypotony in Rabbits' Eyes.**

The PRESIDENT followed with a paper on this subject. He said the intraocular tension was lowered by (1) paracentesis of the anterior chamber; (2) trephining the vitreous chamber. Eyes so operated upon were excised half an hour and an hour later, and then hardened in Zenker's solution. A serous effusion into the interior of eyes so hardened shows as a granular coagulum. In the eyes in which the aqueous humor was evacuated this granular coagulum was found completely filling the anterior chamber, but did not pass backwards through the pupil: though there was some found in the periphery of the posterior chamber about the anterior part of the ciliary body. In the eyes in which the vitreous was trephined, granular coagulum was found, filling the anterior chamber; in the vitreous extending back from the posterior part of the ciliary body; raising the epithelium of the anterior part of the ciliary body in the form of blisters; on the inner surface of the retina at the posterior pole, and in the supra-choroidal lymph spaces. He thought there could be little doubt that the exudate external to the choroid and on the inner surface of the retina came from the veins of those structures, owing to the alteration of the relation of blood-pressure to intraocular pressure. In the same way, the serous exudate in the anterior chamber probably came from the veins of the iris. There was no continuity between the clot in the anterior chamber and that in the neighborhood of the ciliary processes, as would be expected if the serous exudate came from the ciliary body. Under normal conditions of intraocular tension the veins of the iris acted as excretory channels for the aqueous humor; when the tension becomes diminished, a serous exudation proceeds from them instead.

Lieut.-Col. ELLIOT agreed that the abundance of the exudate in any part of the eye was an evidence of the difference be-

tween the pressure within and the pressure without the vessels at that point, despite a recent French article which denied there was any circulation within the eye. The contribution of the President he regarded as of great importance.

Sir GEORGE BERRY (Edinburgh) joined in congratulating the author, and said it would be of great interest to know, now, what happened when the ocular tension was gradually reduced, as in the gradual softening associated with interstitial keratitis.

Mr. GEORGE YOUNG maintained that it was not necessary to enter the anterior chamber when operating for chronic glaucoma, and described the double sclerotomy he did, as outlined by him in New York in 1912.

Dr. G. MACKAY (Edinburgh) thought passing through the ciliary body, as presumably Mr. Young did, must injure its secreting function.

The PRESIDENT replied.

Lieut.-Col. R. H. ELLIOT, I. M. S., read a paper on a **contribution to the histology of the trephined disk**. He said that for some time he had been examining the trephined disk. To get the best results, the disks must be cut strictly in the meridional plane of the eye, otherwise the most erroneous conclusions might be drawn. For a trephining operation to succeed, it was probably essential that a portion of Descemet's membrane, or of the pectinate ligament, or both, should be completely removed. In every glaucomatous case which had been examined, there were hyaline thickenings on Descemet's membrane, close to where it was about to break up to form the pectinate ligament, and always on the posterior surface, hence he concluded they were products of the activity of the endothelial layer of Descemet's membrane. Possibly their occurrence bore no relationship to the glaucomatous process, but they might be a result of the chronic congestion attending long-continued high pressure in the eye.

Capt. THOMSON HENDERSON regarded Descemet's membrane as a deposit due to the activity of the epithelium lining the anterior chamber. The foetal eye, in both man and animals, had a very inconspicuous Descemet's membrane, while it was larger the older the owner. He considered that the anatomical cause of glaucoma was the fibrous degeneration of the cribiform ligament.

The PRESIDENT agreed there must be a sound anatomical basis for a proper understanding of the physiology. Only in one zone of the circumference of the eye could a true filtering scar be secured, and that was, as Col. Elliot had pointed out, at the sclero-corneal margin. The operator must keep well forward, and aim at getting a complete circle of Descemet's membrane.

Capt. R. R. CRUISE contended that it was unnecessary, in order to secure a good filtering scar, to excise a circular portion of Descemet's membrane. He had derived better results from a modification of Fergus's sclerotomy flaps, by which incision of Descemet's membrane was avoided.

Mr. HOSFORD agreed with Mr. Cruise and said he and Mr. Brooksbank James had been doing a modified Lagrange.

Mr. GEORGE THOMPSON described four cases of **herpes zoster affecting the ciliary nerves**, and the communication was discussed by several members.

Capt. R. R. CRUISE exhibited a greatly improved design of visor for the Army, the outcome of a careful analysis of the criticisms evolved as a result of hard wear and tear of the pattern submitted to last year's Congress, when the Society passed a strong endorsement of visors being supplied, as thereby a large proportion of eye injuries at the Front could be avoided. The present design he regards as quite fool-proof. A central strut keeps the chain mail away from the nose, the mail is preserved motionless in front of the eyes, and when not in use can be put on to the top of the steel helmet in one second. The shooting accuracy of the wearer was not impaired, and in a bright light it was an advantage.

Col. LISTER agreed that the submitted model was a beautiful one, and there was a surprising apathy about its universal supply. He thought there might be an objection to wearing it with the gas-mask, especially in hot weather.

Mr. GEORGE YOUNG gave a contribution on clinical tests for the threshold of light and color. His plan was to place circles of color of definite degrees of intensity on the leaves of a small album. These leaves are then rapidly turned in view of the patient. He had noted, as a result of many observations by this quick method, a definite relationship between the color-perception and certain diseases. For example, the light

sense was much affected, while in the retinitis of pregnancy the perception of yellow was very much reduced.

Dr. W. W. SINCLAIR spoke highly of the album, which for quick diagnosis, especially of scotomata, was the best thing he had yet encountered.

The afternoon session was devoted to a demonstration and discussion on **plastic operations on the eyelids**. The major part in the event was taken by Maj. GILLIES, R.A.M.C., who has been very successful in his efforts to increase the comfort and appearance of these victims of the war. He not only exhibited a number of soldiers from the special hospital at Sidcup, but demonstrated, by means of the epidiascope, his operative plans, and the successive stages by which an approach to the normal appearance was reached. Many British and Colonial surgeons were present and contributed to the discussion, at the end of which Maj. Gillies was cordially thanked.

### **Contagious Diseases of the Conjunctiva.**

On the following morning, members journeyed to Swanley, to inspect the Ophthalmia School, which is under the direction of the Metropolitan Asylums Board, with Mr. Treacher Collins as Consulting Ophthalmologist. Here a discussion was held on the subject of contagious diseases of the conjunctiva. It was initiated by the reading (by Col. Lister, A.M.S.) of a joint communication by Maj. J. F. CUNNINGHAM and Capt. J. WHARTON. The authors stated that the question of trachoma and conjunctivitis of the ordinary kind arose in consequence of the introduction of colored units into France. 19% of the Egyptians brought into France had active trachoma, and 9% of the Chinese had it in that form. Infected cases were separated from the clean, and kept isolated from them. The authors had no knowledge, at the time they wrote, of any cases in which the disease had spread either to the civilian population or to our own troops. The infected cases were improving under treatment, and the sickness-hours among them did not exceed those in clean Companies. The importation of cases of trachomatous granulations or acute conjunctivitis had been stopped. The object of treatment was not so much cure as to keep the men fit and to prevent the spread of the disease.

Camps for both Chinese and Egyptian Labor Companies were enclosed, and the men not allowed out of their camps, except for work. On no account was a man transferred from an infected to a clean Company. Less than one man per month had had to be repatriated in consequence of the disease. The task of inspection and placing the men into their proper categories was very heavy, and involved traveling many hundreds of miles. This work was admittedly done very efficiently. The universal drop treatment was that carried out; once a day the men were formed into rows, squatted down and drew down their lower lids while looking upwards, and a native orderly passed along administering two drops of the following preparation: acid bor. grs. X., zn. sulph. grs. II, water to 1 oz. In this way a whole Company could be treated in about twenty minutes.

Col. LISTER said he could testify to the splendid work which Maj. Cunningham had done in this connection. If the men coming in had not been properly classified to begin with, the whole scheme would have been wrecked.

The PRESIDENT gave a short account of the work at this School. All children were admitted who had a discharge from the eyes capable of being conveyed to the eyes of another child. Of the 7163 children who had been admitted since 1903, 1697 were diagnosed as suffering from trachoma, and 5466 from other contagious eye affections. In the school, the trachoma cases are kept separate from the others. The causative factor seemed to be an ultra-microscopic organism which did not seem to be transferred through the air, but in moist discharge conveyed from one person to another. Exacerbation of symptoms in cases of trachoma seemed to be always associated with the presence of some well-known bacillus, such as the gonococcus. In trachoma there was an enlargement of lymphoid follicles, due to a chemical irritant, an invasion of the subepithelial tissues, and a new formation of lymphoid tissue. The reason trachoma was not met with anywhere except in the loose subepithelial areolar tissue he regarded as due to the peculiarity of that arrangement. When the areolar tissue was replaced by fibrous tissue, the disease was arrested.

MR. TYRRELL (Medical Officer of the Schools) discussed the

disease and demonstrated treatment of it by means of CO<sub>2</sub> snow.

Mr. M. S. MAYOU gave his view of the histology of trachoma, aided by colored drawings. He mentioned with high approval the use of jequeritol for this disease. The subject was also discussed by Mr. J. B. STORY (Dublin), Lieut.-Col. ELLIOT, Maj. DERBY (U. S. Army), and Mr. GIRI.

The proceedings terminated with two clinical meetings, one at the Royal Society of Medicine, and the other at the National Hospital for the Paralyzed, at both of which many cases of much interest were demonstrated and discussed.

A Museum was held in an adjacent room during the three days, under the direction of Mr. A. C. HUDSON, and on Thursday evening members and guests dined together.

REPORT OF THE PROCEEDINGS OF THE SECTION  
ON OPHTHALMOLOGY OF THE NEW YORK  
ACADEMY OF MEDICINE.

By JOHN M. WHEELER, M.D., SECRETARY.

MONDAY EVENING, MAY 20, 1918.      MARTIN COHEN, M.D., CHAIRMAN.

**Acute streptothrix infection of conjunctiva**, Drs. KNAPP and DWYER (appears in this issue).

DISCUSSION: Dr. C. H. MAY showed a case of **chronic streptothrix infection of the conjunctiva**. This patient has had many different forms of treatment without improvement, and now has contraction of the conjunctiva and other sequelæ.

Dr. CUTLER said that the appearance of the lower lids in Dr. May's case suggested pemphigus.

Dr. THOS. A. MULCAHY reported a case of **tuberculoma of the iris** with pathological findings.

This case is of interest because of the report of the pathological findings and it also emphasizes the value of the general tuberculin reaction.

CASE.—I. H., aged 3 years, was admitted to the New York Foundling Hospital in April, 1915, and was treated as a feeding case. Her first eye trouble was noticed when she was about one year of age together with swelling of the phalanges.

The writer saw the patient for the first time when she was  $2\frac{1}{2}$  years old. She had photophobia, circumcorneal injection; there was a small yellowish white mass in the lower outer quadrant between posterior surface of the cornea and the iris. This tumor was spherical in shape, the size of a pea, sharply outlined, with a few blood vessels on the surface. Pupil irregular and pupillary space contained a fibrous exudate; anterior chamber deep.

Four Wassermanns were negative, the last being a provoca-



tive one. Three von Pirquets were made; the last was mildly positive. A general tuberculin test was positive both locally and generally. The patient died of pneumonia following measles.

At autopsy, lesions found were double lobar pneumonia, many caseous bronchial lymph nodes, and amyloid liver. The left eye, one finger, and lymph node were examined by Dr. James Ewing of Cornell University. His report as to the eye was as follows: "In the iris and about the ciliary body there is a rich infiltration by lymphocytes, plasma cells, proliferating blood vessels, and in the blood vessels and about them are many polynuclear leucocytes. At several points there are focal collections of lymphocytes. These changes are consistent with a tuberculous origin." No tubercle bacilli were found.

Dr. M. URIBE-TRONCOSO presented a case of **unilateral Argyll-Robertson pupil**. Patient is 35 years old, affected with tabes involving both cervical and lumbar enlargements of the cord; the former probably first according to Dr. Joughlin, who studied the patient in the neurological clinic of the Post-Graduate Hospital. He gave a specific history, the infection being acquired seven years ago, the only treatment he has ever received being six intravenous injections of salvarsan taken at the time of the original infection. There is marked incoördination in the legs and arms and a loss of the sense of "touch" and of the postural sense in the hands which is the cause of the ataxia. Rhomberg sign and definite ataxia in walking are evident. The cranial nerves are unaffected, except the fifth. The right pupil is larger than the left and shows no direct reaction to light, but reacts promptly to convergence. Illumination of this pupil produces consensual reaction of the other. The left pupil reacts well to light and accommodation. No consensual reaction can be observed by the naked eye of the right pupil, but with a loupe the indirect response is unmistakable. Using a very strong light and observing with a loupe a faint contraction of the iris muscle can be detected on direct illumination of the right eye. Vision  $\frac{20}{30}$  in both eyes. Accommodation normal. Fundus normal. Visual fields present a slight contraction for white and restriction for colors.

Unilateral Argyll-Robertson pupil is of comparatively rare

occurrence, two or three cases being observed among a large number of tabetic or syphilitic patients in the neurological clinics. The pathological lesion seems to be a breaking of the reflex path between the nucleus of the oculomotor nerve and the anterior corpora quadrigemina. Generally the direct and the consensual reactions (by illumination of the other eye) are abolished, but there are some cases as those studied by Lutz recently and in the speaker's case in which a consensual response can be obtained in the inactive pupil by the illumination of the good eye. This fact cannot be explained when a second decussation of the optic fibers is not admitted between the corpora quadrigemina and the oculomotor nucleus. In many of the best text books the schemes of the optic pathway show no decussation of these fibers, which run directly from the optic tract to the nucleus of the oculomotor on the same side. Commissural fibers joining both nuclei are pictured. Still it is an anatomically proved fact that this second decussation exists, but probably it is only partial.

Dr. URIBE-TRONCOSO presented a case of **tumor of the sphenoid with ocular manifestations**, a girl 11 years old, who when first seen in March, 1918, complained of loss of vision in the left eye. Trouble began about one year ago when after severe headache the mother noticed the eye was "crossed." The school nurse took her to a clinic where she was refracted but without any avail. Family history negative. When seen in March there was a marked divergent strabismus of the left eye. Slight exophthalmos. The root of the nose on the same side is enlarged laterally and slightly prominent. No restriction in the movements of the eye. On palpation a hard bony mass could be felt at the upper and inner angle of the orbit. Vision reduced to light perception. Ophthalmoscopic examination of the left eye showed a complete postneuritic atrophy of the optic nerve. The right eye had  $\frac{1}{2}\frac{5}{8}$  vision and fundus examination showed a mild optic neuritis. Visual field was markedly contracted in a concentric way for white and colors, and the contraction was specially noticeable upward and outward. Wassermann and von Pirquet tests were negative. X-ray examination showed the whole floor of the frontal fossa thickened, with marked encroachment of the left nasal fossa. Marked density in the frontal sinus region and the left eth-

moid and specially the sphenoid sinus. On lateral posture a marked sclerosis is apparent in the region of the sphenoid sinus. The sella turcica is small.

The author thinks this sclerosing process is due to a new growth, of comparatively benign course, it having lasted for about one year. It is almost certainly an osteoma of the eburnous type, since osteo-sarcomata of this region are very malignant especially in children. It is very difficult to say when the tumor began, but all data point to a former invasion of the sphenoid bone and the eye findings support this view. The slight exophthalmos indicates that the cavity of the orbit has been reduced only slightly. Compression and inflammation of the optic nerve began first in the left side and ended in blindness; the right eye being spared until lately when the papillitis started. It is a well known fact that tumors of the sphenoid never produce compression of the chiasm before involving the optic nerves. Hemianopsia has never been observed because the chiasm is not directly in contact with the sphenoid bone, being separated from it by the anterior part of the hypophysis. According to Lagrange osteomata of the cranial cavity which reach behind the orbit are exceedingly rare.

Dr. SINCLAIR TOUSEY said that the anteroposterior picture presents an apparent blocking of the nasal fossæ which may be due to the opacity of the body of the sphenoid and not to an extension of the sclerosis to the septum or turbinated bones.

Dr. GUTMAN said it is hard to understand the presence of an osteoma and believes that the condition is better explained by a syphilitic process.

Dr. URIBE-TRONCOSO referred to the well-known fact that an empyema of one sphenoid sinus may affect both nerves. Osteoma may act in the same way. Probably the condition is not luetic as the patient is a small child, the Wassermann is negative, and there is no sign of lues.

Dr. L. W. CRIGLER presented two cases of **vernal catarrh of the bulbar type**. One was that of a boy 9 years of age who had had a recurrence every spring for the past five years. His father stated that he was similarly affected for twenty successive years, after which the disease entirely disappeared. The

second case was that of a girl, age 7. She had had three successive recurrences.

The cases were presented because of their striking similarity to the text-book description of the disease, being confined exclusively to the eyeball, and for the further purpose of securing an expression of opinion from the members of the section regarding treatment.

Asked for some remarks upon radium treatment for vernal catarrh, Dr. Tousey said it is wonderfully effective but the treatments have had to be repeated three or four times a year in the cases so far treated by him. He always protects the eyeball by a concave polished metal shield, which is self-retaining in the conjunctival sac. This is to avoid any possibility of lessening the transparency of the cornea or lessening the sensitiveness of the retina. Asked whether he had ever seen such an effect from radium he said that he had not, but from analogy with the X-ray which presents such a danger, he always shields the eyeball when treating only the lids. Asked in regard to treating diseases of the eyeball itself by direct applications of radium, he said he should not hesitate to do so but would mention to the patient the possibility of the two effects mentioned and should shield every part of the eyeball where the curative effect was not required.

Dr. CUTLER had not seen injurious results from the use of radium in vernal catarrh except a slight haziness of the cornea which may have been due to radium exposure. In a case of glaucoma that refused operation, the affected eye had three exposures to radium. There is some reduction of tension and Dr. Cutler thinks something may be promised by this treatment. He had seen loss of eyelashes from radium exposures.

Dr. MAY had a patient who had radium exposures without protection of the globe. Over a year has elapsed without sign of injury from exposure.

Dr. MCDANNALD had a case which was exposed and relieved of symptoms and the lids were cured of elevations, but irritation followed exposures and patient's vision is slightly reduced as a result.

Dr. REESE had a case of epibulbar epithelioma which yielded to radium after the vessels leading to it had been tied off.

There was no protection of the globe and no impairment of vision resulted.

Dr. W. F. C. STEINBUGLER presented a case of **retinitis circinata**. S. J., female, 57; family and previous history negative. Wassermann negative. Urine negative. Blood pressure 100. Came to the Knapp Hospital clinic April 16th of this year complaining of itching of eyes for some time and poor central vision of the right eye for the last six months. V. R.  $\frac{4}{200}$ ; L.  $\frac{20}{50}$ . There is a moderate degree of chronic conjunctivitis. On examining the fundus of the right eye a reddish-brown spot, slightly larger than the disk, is seen in the macular region, surrounded by a horse-shoe shaped white patch. The long diameter of the horse-shoe measures about three times the breadth of the disk and lies with its long axis transversely, the convexity of the horse-shoe pointing towards the disk and being very close to it. There are many small white patches in the neighborhood of this figure, scattered both above and below, and at the temporal end are several small shining spots which resemble crystals of cholesterolin. The temporal blood vessels can be seen near the margins both above and below. The vessels are not tortuous. The fundus of the left eye is normal.

Dr. WALTER D. EDWARDS reported a case of **acute retrobulbar neuritis of obscure origin associated with tooth infection**. Mrs. R. N., age 38, was first seen April 15, 1918, at the Knapp Memorial Eye Hospital complaining of sudden loss of vision of left eye and slight pain in moving the eyeball. The vision was reduced to hand movements, eccentrically, and there was a well defined central scotoma. The fundus examination was negative, except for engorgement of the retinal veins. The sinuses were reported negative after two examinations and the radiographs showed nothing definite. Examination of the blood and urine was also negative. The teeth were radiographed and a large area of infection about the roots of the upper first left molar was disclosed. This tooth and three others which showed poor root fillings were extracted. Two days later the symptoms became much worse—hemorrhages were noted on the disk, which was swollen 2 D. Strabismus developed, the eye was turned up and out, and the vision was reduced to hand motion in the extreme temporal field. The

condition gradually subsided until at the time of this report the vision is  $\frac{18}{200}$ . There is still an absolute central scotoma for colors and a relative central scotoma for form. The optic disk shows beginning atrophy on the temporal side.

The case is of interest as being an acute retrobulbar neuritis with marked ophthalmoscopic changes and orbital symptoms (paralysis of several muscles) associated with a distinct tooth infection. The extraction of the affected teeth was followed by only a partial recovery. The case will be kept under observation and will be again reported upon later.

Dr. GEO. H. BELL presented a woman, age 60, with an extensive orbital tumor which had been treated with radium. The X-ray plate showed the orbit to be filled by a growth and there was a soft looking area in the frontal bone above the orbit. The tumor in the orbit and on the brow has been absorbed. The patient was treated by Dr. Sinclair Tousey with radium. Dr. Bell exhibited several photographs, showing the marked improvement in the patient's condition. The cosmetic result is now almost perfect.

DISCUSSION by Dr. SINCLAIR TOUSEY. The patient came with her whole head enveloped in a black veil on removing which she presented a ghastly spectacle. The lower eyelid was greatly stretched and so was the eyebrow and the skin of the forehead. The size of the tumor is evidenced by the fact that the radium was applied to six different areas separated by  $1\frac{1}{2}$  inch of untreated surface. The radium instrument was in contact with an area about  $\frac{1}{2} \times \frac{3}{4}$  inch and the effect of course extended in every direction. And as only the very penetrating rays were employed the expectation was that every part of the tumor would be equally affected. Twenty milligrams of radium salt having a radio activity of two million, *i.e.* 20 millicuries, were used. The radium salt was in a sealed glass tube and that was covered by sheet lead and then by thick sheet rubber. The function of the latter was partly to prevent contamination of the radium instrument through contact with the patient, and partly to arrest secondary rays arising from the lead where the penetrating rays emerged. These secondary rays have little penetration and would be absorbed by the skin and produce a dermatitis during long exposures. Each treatment consisted of an application of one

hour to only a single area. The first application was made to the outer lower part of the eyelid. A week later one was made to the inner lower part of the lower eyelid. Other areas treated in successive weeks were the outer part of the eyebrow, the inner part of the eyebrow, the upper part of the forehead far to the left, and the upper part of the forehead near the median line. The first treatment was on December 19, 1917. By December 31st the tumor had decidedly diminished in size and the skin over it was shriveled. The reduction in size was progressive and a smaller number of areas were treated so that each of the last two remaining areas received a treatment every other week as compared with the one treatment in six weeks given to each area at the beginning. The last treatment was given April 1, 1918, three months and a half after the first, and now two months later not a trace of the tumor remains and the patient seems perfectly well. At no time was there redness or inflammation of the skin from the radium applications.

REPORT ON THE PROGRESS OF OPHTHALMOLOGY  
FOR THE SECOND, THIRD, AND FOURTH  
QUARTERS, 1917.

By H. KOELLNER, Berlin; W. KRAUSS, Marburg; R. KÜMMELL, Erlangen;  
W. LOEHLEIN, Greifswald; H. MEYER, Brandenburg; W. NICOLAI,  
Berlin; H. PAGENSTECHER, Strassburg; K. WESSELY, Würzburg;  
and M. WOLFRUM, Leipsic; with the Assistance of Drs. ALLING, New  
Haven; CALDERARO, Rome; CAUSÉ, Mayence; CURRAN, Kansas City;  
DANIS, Brussels; GILBERT, Munich; GROENHOLM, Helsingfors; v.  
POPPEN, Petrograd; TREUTLER, Dresden; and VISSER, Amsterdam.

Edited by MATTHIAS LANCKTON FOSTER, M.D., F.A.C.S.,  
New Rochelle, N. Y.

(Concluded)

IX.—THE LIDS.

57. GUÉNOD, A. A contribution to the study of the pathogenesis and treatment of marginal blepharitis. *Clinique ophthalmologique*, viii., 4.  
58. MADDOX, E. E. A new operation for ptosis. *British Journal of Ophthalmology*, June, 1917.  
59. MORAX, V. A new technique for the treatment of total symblepharon. *Annales d'oculistique*, cliv., 6.

In three cases with a complete obliteration of the conjunctival sacs and adhesions of the two lids by an extensive orbitopalpebral scar MORAX (59, **New operation for total symblepharon**) has succeeded in reestablishing the interpalpebral fissure and a small socket for a prothesis by employing the following technic:

*First Step.* (1) A horizontal incision 4 to 5mm long and 4 to 5mm deep is made through the skin and subcutaneous tissue all along the interpalpebral fissure. (2) Two vertical incisions are drawn at the extremities of the horizontal one,



extending above to the eyebrow and below to the inferior level of the lower lid. The depth of these two incisions is the same as that of the horizontal one. When finished the incisions have the shape of an H. The flaps are carefully dissected up, including as much of the fibers of the orbicularis muscle as possible. The upper flap is turned upwards and its margin is sewed by three sutures to the skin. The lower flap is turned downwards and also fixed to the skin of the face by three sutures. In this manner the raw surface of the flaps remains exposed and at this stage it is covered with large grafts of epidermis, taken from the arm, thigh, or abdomen. A layer of oiled silk is applied directly on the grafted flaps and the wound is bandaged. The wound is inspected after three or four days and the sutures removed on the fifth day.

*Second Step.* About fifteen days later, when the reaction has subsided, the adherent upper margin of the upper flap and lower margin of the lower flap are freed and the flaps are returned into primary position. A shell of lead or glass is introduced into the space corresponding to the orbital cavity and the free margins of the flaps are sewn together above the shell. The flaps are left in this condition for about two or three months. After this interval, when there is a minimum risk of further retraction of the tissues, the *third and last step* is made. This consists of a horizontal incision, separating the two flaps. Forty-eight hours later the shell is removed and replaced by a prothesis. If the flaps are too soft, they can be strengthened by the transplantation of a piece of cartilage taken from the ear.

SCHOENBERG.

GUÉNOD (57, *Pathogenesis and treatment of marginal blepharitis*) thinks that the pathological substratum of the predisposition of certain individuals to a chronic blepharitis consists of a lack of elasticity of the capillaries of the margins of the lids, and of the Meibomian and sebaceous glands. These individuals have a tendency also to varicosities of the veins and of the lymphatics in other parts of the body (hemorrhoids, lymphatic varicosities, varicocele, varicosities of the veins of the legs, etc.). In such patients any slight irritation of the lids or conjunctiva may produce a capillary congestion which fails to recover and becomes chronic. Very frequently

the hair follicle becomes the prey of the staphylococcus aureus, which is always found on the skin of the lids, and ulcerative blepharitis is the final outcome. The author recommends for chronic congestion of the margins of the lids the frequent use of adrenalin drops in the conjunctival sac, as a stimulant for the capillary contraction. For the ulcerative form he performs a complete epilation of all the ciliae, applies carefully an iodine-acetone solution on the margin of the lids, and bandages the eye for twenty-four hours with gauze moistened with glycerine. He repeats the treatment in case of recurrence.

SCHOENBERG.

MADDOX (58, **A new operation for ptosis**) points out the disadvantages of the usual operations for ptosis which approach the defect from the skin side of the lid. He suggests a modification of a procedure practiced by Sir William Bowman. The lid is double everted and the levator tendon exposed. The tendon is lightly cauterized, and a supporting tendon passed. The apex of the tarsus is snipped off, and the two ends of the suture passed through it. It is almost impossible from the description to gather exactly how the sutures are inserted. There is a figure, but it is not sufficiently elaborated. Maddox has practiced the operation for four or five years, and has been gratified by the results obtained.

T. HARRISON BUTLER.

#### X.—THE LACRIMAL ORGANS.

60. FAVA, A. **A rare affection of the lacrimal apparatus.** *Annales d'oculistique*, cliv., 6.

61. JOCQS. **The lacrimal tumor.** *Clinique ophthalmologique*, viii., 4.

62. PETIT, P. **A few words on the technique of extirpation of the accessory lacrimal gland.** *Annales d'oculistique*, cliii., 11.

FAVA (60, **A rare affection of the lacrimal apparatus**) reports the case of a young peasant who had several subcutaneous abscesses of the face, an ulcer in the region of the lacrimal sac, extending deep down into the sac, and three other ulcers in the inferior maxillary region of the left side. The preauricular and latero-cervical lymphatic glands were enlarged. Cultures made on gelatine containing glucose and glycerine, as well as injections in guinea pigs, revealed the presence of nocardia, a

filamentous microorganism belonging to the same species as streptothrix.

SCHOENBERG:

JOCQS (61, **The lacrimal tumor**) repeats his belief in the extirpation of the mucous membrane of the lacrimal sac by cauterization with a 50% solution of zinc chloride as the most efficient method in the treatment of dacryocystitis.

SCHOENBERG.

PETIT (62, **A few words on the technic of extirpation of the accessory lacrimal gland**) suggests the introduction of a suture through the outer third of the palpebral conjunctiva of the upper lid, in order to exert a continuous traction on the subconjunctival tissue and facilitate the appearance of the gland. It is well to begin the excision of the glandular lobules at the internal portion, which is much less vascular than the external portion.

SCHOENBERG.

#### XI.—ORBITS AND ACCESSORY SINUSES.

63. CARLOTTI. **Enucleation with transplantation of costal cartilage in the orbital cavity.** *Annales d'oculistique*, cliii., 4.

64. FROMAGET, H., and BERTEMES, G. **Pseudoconjunctivitis accompanying fronto-ethmoidal sinusitis.** *Ibid.*, cliii., 8.

65. KIRKPATRICK, MAJOR B. **Double orbital sarcoma.** *British Journal of Ophthalmology*, June, 1917.

CARLOTTI (63, **Enucleation with transplantation of costal cartilage in the orbital cavity**) publishes ten cases of this nature. In six of the cases he employed cartilage with skin, in the last four cases he used only cartilage and the results are most satisfactory. He advises to suture the two horizontal muscles and the superior and inferior recti on top of the cartilage and then to sew up the conjunctiva.

SCHOENBERG.

FROMAGET and BERTEMES (64, **Pseudoconjunctivitis accompanying fronto-ethmoidal sinusitis**) report a number of cases of this condition. It is usually unilateral and consists of a congestion of the bulbar as well as palpebral conjunctiva. There is very little or no mucous secretion; lacrimation is very persistent. The skin of the lids is not irritated, as it usually

appears in artificially induced conjunctivitis. The patients usually complain only of a slight burning in the eyes. The duration is of from one to two weeks. There is a tendency of recurrence every three or four months. The ætiology is easily established by a rhinological examination. Pressure upon the floor of the frontal sinus on the same side with the conjunctival affection elicits a sharp pain. This is characteristic of a frontal sinusitis. The patient is usually not aware of his nasal trouble. The "conjunctival crises," as we could term the occasional attacks of congestion in the eye, coincide with a recrudescence of the fronto-ethmoidal sinusitis, probably due to a stoppage of drainage. Discussing the pathogenesis of these conjunctival reactions they think that the inflammatory process extends from the sinuses along the anterior and posterior ethmoidal, suborbital, and nasofrontal veins to the venous plexus of the orbit.

SCHOENBERG.

The paper of KIRKPATRICK (65, **Double orbital sarcoma**) describing this tragic case is illustrated with a photograph of the patient, and with microphotographs of the sections of the tumor, which was an endothelioma of sarcomatous type.

T. HARRISON BUTLER.

## XII.—THE CONJUNCTIVA.

66. AUBINEAU, E. Remarks on artificially induced conjunctivitis. *Annales d'oculistique*, cliii., 8.

67. BUTLER, T. H. Some remarks upon spring catarrh: with special reference to its diagnosis and its treatment with radium. *British Journal of Ophthalmology*, July, 1917.

68. COSSE and DELORD. Cases of conjunctivitis in dysenterics. *Annales d'oculistique*, cliv., 1.

69. COSSE and DELORD. Ipecacuanha conjunctivitis. Its microscopic diagnosis. *Ibid.*, cliv., 3.

70. FROMAGET and HARRIET. Conjunctivitis and pseudo-trachoma artificially produced by soldiers by the introduction of emetin into the conjunctival sac. *Ibid.*, cliii., 9.

71. GIFFORD, H. On the frequency of occlusion of the inner end of the canaliculus in old trachoma. *Ophthalmic Record*, September, 1917.

72. GOLDBACH, L. J. Lymphatic nodular keratoconjunctivitis. *Journal of the American Medical Association*, July 14, 1917.

73. GOLDENBERG, M. The pathogenesis of ophthalmia eczematosa. *Ibid.*, July 14, 1917.

74. HARDY, W. F., and LAMB, H. D. Essential shrinking of the conjunctiva with report of two cases. *American Journal of Ophthalmology*, October, 1917.

75. KALT, M. Method of discovering the presence of powdered ipecacuanha in the conjunctiva. *Annales d'oculistique*, cliii., 6.

76. LAMB, F. W. Conjunctivitis tularensis (squirrel-plague conjunctivitis), with report of a case. *Ophthalmic Record*, May, 1917.

77. LAWSON, A. Three cases of infection of the conjunctiva from the fur of cats. *British Journal of Ophthalmology*, May, 1917.

78. LEIPER, R. T. Thelaziasis in man: a summary of recent reports on "circumcorneal filariasis" in Chinese literature, with a note upon the zoölogical position of the parasite. *Ibid.*

79. MOREAU, F. Electric ophthalmia. *Annales d'oculistique*, cliii., 8.

80. STUCKEY, E. J. Circumocular filariasis. *British Journal of Ophthalmology*, September, 1917.

81. WHITE, D. W. The new incision and crushing operation for advanced trachoma. *Ophthalmic Record*, May, 1917.

E. AUBINEAU (66, **Remarks on artificially induced conjunctivitis**) warns the reader to be on the lookout when examining soldiers with a unilateral conjunctivitis and mostly if the process is localized only on one lid.

COSSE and DELORD (69, **Ipecacuanha conjunctivitis**) have seen several soldiers with conjunctivitis induced by the introduction of powder of ipecacuanha, pepper, or tobacco. The conjunctivitis is usually limited to the lower lid; microorganisms are never present and there is no secretion of mucus. There are no corneal complications. The condition clears up in a few days if the eye is sealed up by a collodion bandage. They describe the microscopic appearance of the granules of various substances found in these forms of induced conjunctivitis and warn ophthalmologists to be on the lookout for malingerers who have escaped active service at the front for many months by the induction of artificial conjunctivitis.

SCHOENBERG.

FROMAGET and HARRIET (70, **Conjunctivitis and pseudo-trachoma artificially produced by soldiers by the introduction of emetin into the conjunctival sac**) say that the inflammation is usually unilateral, mostly in the right eye; the skin of the lids is irritated; there is no discharge of mucus or pus. The conjunctiva has a salmon color and it often has the appearance of trachoma, but the cornea is never involved. The condition clears up in about one week if no new supply of ipecac powder

is introduced. In doubtful cases, where trachoma is suspected, the sealing up of the eye by a collodion bandage for several days will establish the diagnosis. In order to establish whether the powder of ipecac acts as a foreign body or as a chemical substance the authors introduced in the conjunctival sac of a soldier powder of ipecac, of another a few drops of an extract of ipecac, and of a third a few drops of a 2% solution of emetin hydrochloride. In all three of them the symptoms of irritation appeared in from ten to twenty hours later, which fact proves, according to the author, that the action of ipecac on the conjunctiva is of a chemical nature.

SCHOENBERG.

Some soldiers in the French army have introduced powder of ipecac into the conjunctival sac in order to produce a conjunctivitis which may dispense them from active service. KALT (75, **Method of discovering the presence of this powder in the conjunctiva**) employs the following method for detection. A small swab of gun cotton is slightly rubbed on the conjunctiva, caruncle, and margins of the lids, then dried in the incubator at 50°, and dissolved in a few drops of alcohol and ether, 1 to 2. After the mixture has evaporated, a few drops of water are added and it is left again to dry. The pellicle which remains is spread on a slide with a drop of water and examined under the microscope. The ipecac appears as dark masses which turn blue by addition of Lugol's solution, showing the presence of starch.

SCHOENBERG.

COSSE and DELORD (68, **Conjunctivitis dysenterics**) say that this complication has never been reported before and that they have seen it in twelve patients. In four of them the conjunctivitis appeared on the fifth to the twelfth day after the onset of the disease and cleared up in from six to seven days; in eight it appeared at the same time with a polyarthritides of the knee, elbow, tibio-tarsal, and shoulder. In one patient the conjunctivitis preceded the dysentery. The conjunctiva was moderately inflamed; the mucous discharge was scanty and contained no microorganisms. The eye regained its normal appearance in ten days. One single patient developed iritis and marginal keratitis of the right eye. The conjunctivitis was more severe in patients with complications in the joints.

As to the etiology, the authors believe that the conjunctival complication is due to toxæmia and not to microorganisms, since repeated microscopic examinations always proved negative, and the clinical evolution was entirely different from that seen in bacterial infections.

SCHOENBERG.

LAMB (76, **Squirrel-plague conjunctivitis**) reports the case of a girl who after preparing rabbits for a meal developed œdema of the lids of the left eye with ulcers on the palpebral conjunctiva and tender preauricular and cervical glands. The proper diagnosis was made and the bacillus recovered. Inoculation of guinea-pigs with secretion from the conjunctiva proved fatal. The bacillus was grown on egg-yolk medium. Treatment with autogenous vaccines seemed to have had a marked effect for the case recovered in about a month, whereas the progress of the two other cases reported was very tedious.

ALLING.

LAWSON (77, **Infection of the conjunctiva from the fur of cats**) cites three cases in which infection of the conjunctiva in children was caused by fondling cats. There seems little reason to doubt that all animals are dangerous pets for small children. The cat is probably the worst because she is more likely to be fondled.

T. HARRISON BUTLER.

In one of MOREAU's two cases (79, **Electric ophthalmia**) there was a period of incubation of eight hours between the exposure to the strong electric light and the conjunctivitis. In both there was a loss of superficial layers of corneal epithelium as shown by fluorescein. The loss of substance occupied a horizontal zone corresponding to the interpalpebral space, directly exposed to the lights. The condition cleared up in from thirty-six to forty-eight hours.

SCHOENBERG.

The disease is rare in England. BUTLER (67, **Spring catarrh**) sees about one case in two years, which is an incidence of 1:10,000. The author laid stress upon the constant presence of an excess of eosinophil cells in a smear, a sign which he regarded as pathognomonic of the disease. The clinical features are characteristic, and a surgeon who has seen a typical case will not readily overlook another. It is easier to make the

reverse mistake, to see spring catarrh when it is not present. Unusual cases of phlyctenular conjunctivitis may cause this confusion, but the presence or absence of eosinophil cells will at once clear up any doubt.

It should not be mistaken for trachoma, but when the two diseases co-exist, as they do in the Near East, a mistake can be made.

The author showed the Congress a case which had been greatly improved by radium, and which was ultimately cured. He cited another bad case which had also lost all symptoms of the disease after radium treatment, but in this patient a troublesome conjunctivitis gave trouble for some long time.

Butler regards radium as the only agent which has any influence upon the disease, and he quotes Sir J. Mackenzie Davidson, who states that in every case without exception which he has treated with radium the plaques have completely disappeared without leaving any scars. He looks upon radium as a specific for the disease.

T. HARRISON BUTLER.

GOLDENBERG (73, **Pathogenesis of ophthalmia eczematosa**) is of the opinion that phlyctenulosis is not a tuberculous manifestation but is due to intestinal putrefaction. He finds that nearly all the patients consume an excessive amount of carbohydrates in the form of sweets. He got the best results in treatment by restricting the diet and administering calomel while treating the eyes locally with atropine.

ALLING.

GOLDBACH (72, **Lymphatic nodular kerato-conjunctivitis**) believes that phlyctenules are strongly suggestive of tuberculosis—if not, then certainly something closely associated with it. In seven of the thirty-nine cases studied, there were signs of active pulmonary tuberculosis. Sixteen had some form of tuberculosis, such as adenitis or tuberculous bone lesions; thirty-two had positive von Pirquet. He advocates prolonged treatment with tuberculin.

ALLING.

GIFFORD (71, **Occlusion of the inner end of the canaliculus in old trachoma**) thinks that most of the cases of trachoma suffer from this complication and has frequently found pus pockets in the canaliculus. This condition should be looked



for, especially before operation, and the canals slit and kept open.

ALLING.

The first case of HARDY and LAMB (74, **Essential shrinking of the conjunctiva**) was a young man of 17 whose history shows that a vesicular eruption appeared on the skin when he was nine years old, two months after vaccination. The eyes began to discharge and had a dry grayish appearance. The right eye presented anterior staphyloma and was almost fixed in position by cicatricial bands. There was almost complete obliteration of the fornices. The left eye had vision of 3/96, the cornea being opaque in its lower third and the lower fornix nearly obliterated.

The second case was a colored boy of 11 who had had a skin eruption occurring in the fall for six years. The eye trouble began with one of the attacks, and after two years the vision was reduced to light perception. The conjunctiva was dry and shrunken but not so extensively as the other case, but both corneæ were opaque. This disease occurs once in about twenty thousand eye cases.

ALLING.

STUCKEY (80, **Circumocular filariasis**) reports the case of a Chinese who came to the hospital at Peking complaining that worms came out of his eye.

On examination four white worms were removed from the conjunctival sac. The organism was found to be *Filaria palpebralis*. The disease has been described in the horse and ox.

T. HARRISON BUTLER.

LEIPER (78, **Thelaziasis in man**) deals with filaria in the conjunctival sac as seen in China in man and animals. The author holds that these parasites are probably identical with *Thelazia callipæda* which is a common eye worm with the dog in Central Asia.

T. HARRISON BUTLER.

WHITE (81, **New incision and crushing operation for advanced trachoma**) dissects the conjunctiva free from the tarsus and then the tarsus from the underlying tissue. With a pair of scissors the tarsal plate is cut into numerous vertical strips which are crushed with roller forceps. The conjunctiva is then

sutured back in place. The tarsus shrinks into a thin cicatricial membrane and thus, it is claimed, relieves the cornea from pressure.

ALLING.

### XIII.—THE CORNEA AND SCLERA.

82. BRUNETIÈRE and AMALRIC. Grave hemorrhage following an extensive injury of the sclerotic. Suture. Recovery. *Clinique Ophthalmologique*, viii., 5.

83. BUXTON, L. H. Dumb-bell keratitis. *Ophthalmology*, April, 1917.

84. DERBY, G. S. Interstitial keratitis—with special reference to the end results. *Ophthalmic Record*, November, 1917.

85. MAGITOT, A. Kerato-conjunctivitis accompanied by nasal lesions. *Annales d'Oculistique*, cliii., 6.

86. MAGITOT, A. A critical study of certain biological properties of the corneal tissue and of human keratoplasty. *Ibid.*, cliii., 9 and 10.

87. STEPHENSON, S. A note upon the pseudo-neoplastic form of interstitial keratitis. *British Journal of Ophthalmology*, December, 1917.

88. VERHOEFF, F. H. The treatment of hypopyon keratitis. *Journal of the American Medical Association*, June 30, 1917.

89. WALKER, SYDNEY. Present status of corneal transplantation and some experimental data. *Ophthalmic Record*, August, 1917.

90. WIENER, M. A new operative method for the relief of advanced cases of keratoconus. *Journal of the American Medical Assoc.*, September 8.

BUXTON (83, Dumb-bell keratitis) has seen three cases of corneal ulcer which he calls by the above title. A small ulcer appears about 2mm below the upper sclero-corneal junction, and from this a line extends perpendicularly to a similar ulcer near the lower margin. The process in the two knobs seems to be a destruction of the epithelium down to Bowman's membrane, but only of the superficial epithelium in the connecting line. There is very little local reaction and the ulcers heal in a few weeks. He does not think that the process is allied to dendritic keratitis but is rather neuropathic and of herpetic nature.

ALLING.

DERBY'S (84, Interstitial keratitis—with special reference to the end results) study of ninety-six cases, with a total of 190 eyes, shows the following results: Corneal opacities of every degree of density were found in 168 eyes. Vascularization was absent in only fifteen and seems to be permanent.

It was observed in one case fifty-five years after the original inflammation. Evidence of iritis was found in sixty-two eyes. It would appear however that its occurrence bears no relation to the severity of the keratitis. In four cases slight opacity of the lens was found. Vitreous opacities were present in a few cases and in 55% lesions of the choroid and retina were found. These lesions sometimes antedate the corneal process. Vision in 161 eyes examined was less than 1/10 in twenty-five. Regarding the cases of typical interstitial keratitis the author is of the opinion that they are, with few exceptions, syphilitic. In thirty-seven cases carefully investigated, evidence of recurrences was found. In spite of the discouraging results, antisyphilitic treatment should be thoroughly carried out.

ALLING.

The rare cases described by STEPHENSON (87, **A note upon the pseudo-neoplastic form of interstitial keratitis**) all occur in children affected with congenital syphilis. The appearances are constant and may be described as follows: Occupying the upper or the lower part of the cornea there is a somewhat prominent, fleshy-looking mass, having the appearance of a neoplasm. The cornea adjoining the mass has shown interstitial deposits, or the whole cornea not occupied by the mass has the appearance of aggressive interstitial keratitis. In course of time the mass has flattened down and there has been no tendency to bulging. The cases follow the normal course of interstitial keratitis.

The disease may be confused with a tumor growth. The age of the patient and the presence of syphilis should help the diagnosis.

T. HARRISON BUTLER.

MAGITOT (85, **Kerato-conjunctivitis accompanied by nasal lesions**) has seen a large number of soldiers with this trouble. The examination of one hundred cases of kerato-conjunctivitis by a rhinologist revealed the following percentages of nasal troubles. Acute rhinitis, 30%; atrophic rhinitis, 5%; tertiary lues in the nose, 7%; maxillary or frontal sinusitis, 13%; nasal polypi, 3%; septal deviations, 25%; hypertrophy of inferior turbinates, 15%. The ocular condition presents certain peculiarities: the conjunctiva is congested, but does not

secrete mucus, the cornea is slightly hazy and the limbus shows a few small and superficial ulcerations. The process disappears after a few weeks but reappears later on. The condition is cured only after the nasal trouble is relieved. The author thinks that this type of kerato-conjunctivitis is due to a reflex nervous irritation starting in the nose. He was able to cure it completely only after the patient underwent the proper nasal treatment.

SCHOENBERG.

VERHOEFF (88, **Treatment of hypopyon keratitis**), after reviewing the various means of treating ulcers and demonstrating their ineffectiveness, explains his own procedure. He makes a crucial incision through the base of the ulcer and then allows a highly concentrated Lugol's solution (25% iodine) to lie in contact with the ulcerated area for about five minutes, flushing it out with boric acid solution. In very small ulcers the solution is applied without the incision, but in rapidly progressive cases he thinks the Saemisch incision is necessary. He has treated forty-two cases and in only eight of the severer ones did the method fail to check the process.

ALLING.

MAGITOT (86, **A critical study of certain biologic properties of the corneal tissue and of human keratoplasty**) says that experience has taught us the following practical points: (a) the excision of a portion of a leucomatous cornea is followed by a regeneration of opaque tissue if the area surrounding the excised piece was opaque; (b) the ablation of a leucomatous piece of cornea together with a portion of surrounding healthy tissue may be followed by a regeneration of transparent tissue; (c) if a disk of leucomatous cornea is transplanted on a healthy cornea we find after a few months that the transplanted piece is very much more transparent. Regarding the question of grafting pieces of clear cornea on a leucomatous cornea the author reiterates what he said in previous papers. *Autoplasty*, using cornea from the same patient, has the greatest chance of success, 95%; *homoplasty*, using a piece of cornea of another individual, succeeds in about 60%; *heteroplasty*, the grafting of cornea from a different species, has never succeeded. Besides the origin of the graft, the question of perfect technic and the clinical condition of the cornea of the

patient determine the success or failure of the attempt. Of all the leucomas those due to burns, ulcers, and pterygium are the most suitable for grafting. Experience has shown that leucoma due to trachoma, interstitial keratitis, or pemphigus is not suitable for an operation. Regarding the technic he advises to have the graft much thinner than the opaque piece which is to be replaced, because it swells up as soon as it comes in contact with the tears and projects above the surface of the cornea on which it is grafted, a condition which favors its easy displacement. The author uses a Bowman trephine of about 4 to 5mm diameter and a very sharp angular knife which cut the lamellæ of the cornea with ease and precision. Perfect asepsis is the first requisite for obviating failure. Very gentle handling of the graft avoiding its traumatism is a second requisite. Cocaine 2% instilled three times is sufficient to produce anæsthesia. An overdose of cocaine may injure severely the corneal epithelium. The after treatment is simple. Binocular bandage for forty-eight hours. Then, wet bandage over the operated eye. On sixth day the eyes are left open. The transplanted piece of cornea is slightly opalescent and gradually clears up almost entirely in four weeks. The author has made about forty keratoplasties and almost invariably had success in cases with leucomas due to burns, but failed just as invariably in those due to extensive ulcers. In two cases only he obtained a fair result by excising the opaque area entirely.

SCHOENBERG.

WALKER (89, **Present status of corneal transplantation**), after reviewing the literature, explains the technic which he has developed. He experimented upon dogs by cutting off a layer of the clear cornea from one eye and transplanting and suturing it over the cornea of the other eye, which was prepared by removing a similar piece. He then covered the whole cornea with a conjunctival flap. His first operations were failures because he entered the anterior chamber in making the section or because of the presence of too much blood under the flap. Finally he succeeded in obtaining three good results. He then operated upon three human eyes with leucomata. In the first the flap was not retained. In the second the graft held and only slight cloudiness and organizations were present.

In the third case the graft held and was cloudy but he expected it to clear. No details of the length of time which has elapsed since the operations are given. From his studies he concludes that transplantation of foreign bodies such as glass or of tissue from another species is doomed to failure, but fair results in selected cases may be obtained from grafts taken from the same individual or from another human eye.

ALLING.

WIENER (90, **Operation for keratoconus**) has operated upon both eyes of a patient as follows: he dissected up an elliptical flap of the cornea (8mm by 3mm) near the periphery above and passed sutures through holes in two thin gold plates, then through the edges of the wound. The plates prevent the stitches from tearing through. The flap included the whole thickness of the cornea except Descemet's membrane. The vision before the operation was  $\frac{7}{200}$ ; after  $\frac{18}{200}$ .

BRUNETIÈRE and AMALRIC (82, **Grave hemorrhage following an extensive injury of the sclerotic. Suture. Recovery**) have seen a soldier with a large injury of the sclera measuring 9 to 10mm. It bled very profusely, but healed well after being sutured. The vision gradually improved from H.M. to  $\frac{1}{10}$  and the tension became normal. The authors warn against a precipitated decision in favor of the enucleation of gravely injured eyes.

SCHOENBERG.

#### XIV.—THE IRIS AND CILIARY BODY.

91. DARIER, A. **The treatment of diseases of the iris and of the ciliary body in general.** *Clinique ophtalmologie*, viii., 1.

92. DAVIES, D. L. **Diseases of the eye of obscure origin.** *British Journal of Ophthalmology*, October, 1917.

93. DOR, L. **Anisocoria of bacillary origin.** *Clinique ophtalmologique* viii., 4.

94. LANE, L. A. **Primary progressive atrophy of the iris.** *Ophthalmic Record*, June, 1917.

95. MORAX, V. **Iritis observed during a small epidemic of dysenteriform intestinal infection.** *Annales d'oculistique*, cliv., 1.

96. ROUSSEAU, FERDINAND. **Researches concerning the ætiology and pathogenesis of non-syphilitic iritis.** *Ibid.*, cliii., 4.

97. ZIMMER. **Double metastatic cyclitis following an attack of bubonic plague.** *Clinique ophtalmologique*, viii., 1.

DARIER (91, **Treatment of diseases of the iris**) makes a general review of the present methods of local and general treatment of iritis, iridocyclitis, and iridochoroiditis.

SCHOENBERG.

DOR (93, **Anisocoria of bacillary origin**) has seen several cases of anisocoria in tuberculosis of the apex of one lung. The pupil is dilated in the eye situated on the same side as the involved lung and the refraction becomes hypermetropic 1.25 D. He thinks that this sympathetic irritation mydriasis is characteristic of apical pulmonary tuberculosis just as the Argyll-Robertson pupil is pathognomonic for cerebrospinal syphilis.

SCHOENBERG.

MORAX (95, **Iritis observed during a small epidemic of dysenteriform intestinal infection**) reports two cases in which the infection was not due to either the amœba, or the bacillus of Shiga. Bacteriologists could not establish with accuracy the type of microorganism. The first patient had three attacks of iritis during his illness, each lasting only twenty-four hours.

SCHOENBERG.

FERDINAND ROUSSEAU (96, **Researches on the ætiology and pathogenesis of non-syphilitic iritis**) maintains that non-syphilitic iritis is either a metastatic condition or the first manifestation of an infectious disease. The diagnosis, ætiology, and pathology of non-syphilitic iritis are yet in great obscurity and the problems involved in these great questions are yet to be worked out clinically and experimentally. The clinical study of cases with non-syphilitic iritis shows that some are due to an infectious disease, tuberculosis, gonorrhea, rheumatism, tonsillitis, grip, etc., and others to disturbances of nutrition, as in gout and diabetes. The author thinks that ophthalmologists are not frequently justified to consider as tuberculous those cases with sluggish iritis and iridocyclitis of unknown origin. His researches, histological and by inoculations in rabbits and guinea pigs, have convinced him that they are far from being tuberculous. He is rather inclined to believe that this type of chronic iritis without any manifestations in the rest of the body is due to a pneumococcic infection which localizes itself from the blood in the iris only.

He tried in a series of experiments to produce experimental pneumococcic iritis by first reducing the vitality of the iris, by mechanical, thermal, and chemical injuries through the action upon the vasomotor and trophic nerves of the iris. The conclusions arrived at are that we do not know yet the reasons for the localization of a general infection on the iris. The pneumococcus may cause an experimental iritis even when injected in very small number, if the iris was already the seat of a previous inflammation. Recurrent iritis, if not luetic, is most frequently gonococcal. Acute exudative iritis, occurring during a rhinopharyngeal inflammation, is usually pneumococcal. Chronic iritis is not tuberculous; it has great analogy with sympathetic ophthalmia.

SCHOENBERG.

DAVIES (92, **Diseases of the eye of obscure origin**) emphasizes the now well accepted fact that a diagnosis of rheumatic or gouty iritis no longer satisfies the modern ophthalmic surgeon. He goes on to discuss the various septic sources which give rise to so-called rheumatic affections.

T. HARRISON BUTLER.

LANE'S (94, **Primary progressive atrophy of the iris**) patient, a girl of 20, noticed a small black hole in the iris about two years ago. Later other atrophic areas appeared until the iris was almost completely destroyed. During this time the eye was subject to attacks of inflammation and gradually became considerably reduced in size. The cornea became opaque and a glaucomatous state ensued. After a trephine operation the cornea cleared and vision improved. Later the other eye showed evidences of glaucoma which were relieved by treatment. Increased tension has been recorded in almost all cases previously reported and latent tuberculosis has also generally been suspected. The patient had acne for a number of years and autointoxication may have been a factor.

ALLING.

ZIMMER (97, **Double metastatic cyclitis following an attack of bubonic plague**) reports a case of this nature in which the patient recovered his general health but remained with his vision O. D. = L. P. = O. S. — O.

SCHOENBERG.



XV.—THE LENS.

98. BERNSTEIN, E. J. **An unusually long open wound after cataract extraction.** *Annals of Ophthalmology*, April, 1917.

99. FISHER, J. H. **A note on cataract extraction with a suggestion.** *British Journal of Ophthalmology*, December, 1917.

100. GROS, H., and FROMAGET, H. **Two cases of subchoroidal, expulsive hemorrhages during cataract extraction. Attempt at prophylactic treatment.** *Annales d'oculistique*, cliii., 11.

101. YOUNG, G. **On macular perception in advanced cataract.** *British Journal of Ophthalmology*, June, 1917.

BERNSTEIN (98, **Unusually long open wound after cataract extraction**) operated successfully on a patient 87 years old but on the third day found the lower lid turned in and the wound gaping. The iris was repeatedly prolapsed but could be replaced. After a month he covered the wound with a conjunctival flap but it did not heal. Two months later the wound was not entirely healed and the eye had developed iridocyclitis.

ALLING.

FISHER (99, **A note on cataract extraction with a suggestion**) places a drop or two of a 1% solution of atropine in the conjunctival sac just before he makes the section. Some of the solution enters the anterior chamber, and a rapid and full dilatation of the pupil takes place.

We think that if this method be adopted the surgeon should personally sterilize the solution.

T. HARRISON BUTLER.

GROS and FROMAGET (100, **Two cases of subchoroidal, expulsive hemorrhages during the cataract extraction**) have met with two cases of expulsive hemorrhage following a cataract operation. Believing that this complication is due both to a degenerative condition of the choroidal vessels and sudden reduction of the intraocular pressure, they recommend as a prophylactic measure a preliminary sclerectomy and the use of pilocarpine two weeks before the cataract operation. They practiced this procedure on the second eye of a patient who had lost the first one by an expulsive hemorrhage and obtained a very satisfactory result. The sclerectomy should be performed in the region of the equator. The lowering of the tension prepares the choroidal circulatory system for a further

sudden diminution of the ocular tension occurring during the cataract operation.

SCHOENBERG.

It is difficult to ascertain the soundness of the macular perception in cataract cases, and yet it would be very useful to be able to do so before performing an operation. YOUNG (101, **On macular perception in advanced cataract**) has succeeded in gaining valuable information in these cases by using disks with one, two, and three perforations which are all within a central area of less than three millimeters. These are placed in succession in the trial frame, and the eye is brought near to the frosted focus light of a Thorington's chimney with the diaphragm widely open. The patient is told to look out for the "moons," and then the disks are rotated. An intelligent patient will be able to state the position of the moons. Young thinks that this test would reveal a defective macula.

T. HARRISON BUTLER.

#### XVI.—THE CHOROID.

102. VALENTINE, J. A. **A case of leucosarcoma of the choroid with epithelioma of the lip in the same patient.** *British Journal of Ophthalmology*, September, 1917.

This case is recorded by VALENTINE (102, **A case of leucosarcoma of the choroid with epithelioma of the lip in the same patient**) because of the rarity of two growths of different kinds in the same patient.

T. HARRISON BUTLER.

#### XVII.—SYMPATHETIC OPHTHALMIA.

103. MORAX, V. **Sympathetic ophthalmia.** *Annales d'oculistique*, cliv., 6.

104. WEEKERS, L. **War's lesson on sympathetic ophthalmia.** *Ibid.*, cliv., 4.

MORAX (103, **Sympathetic ophthalmia**) has seen one single case of sympathetic ophthalmia among 1500 soldiers with injuries of the eyes and expresses his entire agreement with Weekers, who, in a paper published on the same subject, states that this disease occurs very rarely. Morax gives the

clinical history and microscopical specimens of the enucleated eye as evidence for the correctness of the diagnosis. The majority of the thirty cases of sympathetic ophthalmia that the author has seen during twenty years have developed, just as the case reported in this paper, after infection following operations on the eye, and he is very much inclined to believe in the infectious nature of this disease. He says that the prompt enucleation of eyes with an iridocyclitis following an infection after a cataract extraction is still the best method of preventing sympathetic ophthalmia as it was in the times of Mackenzie, who was the first to recommend this operation.

SCHOENBERG.

WEEKERS (104, War's lesson on sympathetic ophthalmia) states that according to German authors sympathetic ophthalmia occurred in 55% of the ocular injuries incurred during the Franco-Prussian war of 1870. During the present war, in which the proportion of eye injuries in relation to other injuries is very much larger, sympathetic ophthalmia is almost unknown. The question of the rarity of this complication came up for discussion before the French and British ophthalmologists and the explanation given by most of the authorities was that the rarity of its occurrence is due to two facts: early enucleation and the use of antiseptics. Weekers believes that the German statistics regarding the high frequency of sympathetic ophthalmia are not tenable under our present clinical conception of this condition. Eliminating all the cases with functional disturbances in the non-injured eye, such as lacrimation, photophobia, asthenopia, etc., which have been included hitherto in the statistics, and the number of cases with sympathetic ophthalmia dwindles to a very small percentage even in the statistics of the Franco-Prussian war. Furthermore a careful reading of the papers published on this subject during that period reveals not a single observation in which a typical sympathetic uveitis is described. Taking his own experience with that of others he concludes that sympathetic ophthalmia is a very rare occurrence in peace as well as during war time. He believes that the very rare occurrence of this disease in modern times is due to the treatment promptly applied, to our present-day methods of antiseptics and asepsis, and to our more accurate diagnosis. The conser-

vation of an injured eye is preferable to its enucleation and since sympathetic ophthalmia is to be feared much less than it used to be a conservative attitude is justifiable. Whenever the removal of the eye is indicated he prefers exenteration to enucleation, because a better prothesis can be applied after this procedure.

SCHOENBERG.

#### XVIII.—GLAUCOMA.

105. CLEGG, J. G. Sclerocorneal trephining for hypertony: an experience of two hundred and fifty operations. *British Journal of Ophthalmology*, September, 1917.

106. EWING, A. E. Postciliary scleral trephining for glaucoma. *American Journal of Ophthalmology*, July, 1917.

107. MORAX, V. Secondary glaucoma developing in eyes containing foreign bodies. *Annales d'oculistique*, cliv., 1.

108. SANSUM, W. D. Rapid reduction of intraocular tension in glaucoma. *Journal of the American Medical Association*, June 23, 1917.

The intravenous injection of glucose is known to produce diuresis and dehydration of the tissues, at the same time raising the blood pressure. SANSUM (108, **Rapid reduction of intraocular tension in glaucoma**) experimented upon two cases of glaucoma and found that intraocular tension was lowered under the treatment. The method is susceptible to control and might be of use where it was desirable to produce a rapid decrease in tension preliminary to operation.

ALLING.

MORAX (107, **Secondary glaucoma developing in eyes containing foreign bodies**) has seen, out of 1000 eye injuries, five cases of secondary glaucoma in eyes containing foreign bodies. Literature reports very few similar cases and the author concludes from his own five observations that the presence of a foreign body in the vitreous may give rise to hypertension even without any signs of cyclitis. Furthermore cases of chronic glaucoma following an injury of the eye, in which the media are so changed that an ophthalmoscopic examination is not possible, should have an X-ray study, since his observations show that the presence of a foreign body is not only possible but probable.

SCHOENBERG.

GREY CLEGG (105, **Sclerocorneal trephining for hypertony**) adopts the Elliot method with few modifications. He prefers the Harrison Butler trephine and generally employs a diameter of two millimeters, but removes only the anterior portion of the disk. The conjunctiva is reflected as far as possible but true splitting of the cornea is not advised, for Clegg finds that an aperture covered by corneal tissue heals up solidly. An iridectomy is not essential but is advisable. The conjunctiva is sutured.

The results obtained are as follows, the figures referring to visual acuity:

*Acute Glaucoma:*

Improved	70%
Stationary	25%
Worse	5%

*Sub-Acute Glaucoma:*

Improved	47%
Stationary	42%
Worse	11%

*Simple Chronic Glaucoma:*

Improved	32%
Stationary	48%
Worse	20%

The author notes that some of the bad results were due to the development of cataract which was not traumatic. He observes that if 80% be the true proportion of favorable results in chronic glaucoma it is superior to the 62% claimed for iridectomy.

T. HARRISON BUTLER.

EWING (106, **Postciliary scleral trephining for glaucoma**) suggests trephining the sclera between the superior and external recti muscles. He operated upon a painful eye in a state of absolute glaucoma, and after six weeks there was drainage through the opening and the eye was quiet. Another case of glaucoma simplex with tension 45 with cupped disks and slightly contracted field on the nasal side was similarly

treated and two weeks later showed tension 35 and relief of blurring. In three other cases, one simple glaucoma and two blind aphakic eyes, the operation was performed with satisfactory results. The points in favor of the procedure are the ease and lack of trauma, slow drainage, less liability to hemorrhage, no deformity of the pupil, no risk to the lens protection of the wound by the upper lid, and possible repetition if necessary.

ALLING.

#### XIX.—THE RETINA.

109. BAILLART, P. **The circulation in the retinal artery. An attempt to determine the arterial tension in the branches of the central artery of the retina.** *Annales d'oculistique*, cliv., 5.

110. BARDSLEY, P. C. **The retinal signs of arteriosclerosis compared with those due simply to increased blood pressure.** *British Journal of Ophthalmology*, April, 1917.

111. BLAKE, E. M. **A case of reattachment of the retina. Trephine operation.** *Ophthalmic Record*, November, 1917.

112. FRENKEL, H. **Circular folding of the retina produced by contusion of the posterior segment of the eyeball.** *Annales d'oculistique*, cliv., 1.

113. GRIFFITH, A. H. **Hereditary glioma of the retina.** *British Journal of Ophthalmology*, September, 1917.

114. LAWSON, A. **A case of detachment of the retina at the ora serrata.** *Ibid.*, October, 1917.

115. PRINGLE, J. A. **Multiple aneurysms of the retinal arteries.** *Ibid.*, February, 1917.

116. TAYLOR, F. E., and FLEMMING, B. B. **Bilateral glioma of the retina with multiple metastases.** *Ibid.*, February, 1917.

BLAKE'S (111, **Reattachment of the retina**) case, a girl of 13, had a traumatic detachment with vision reduced to hand movement in the lower part of the field. Ten months after trephining of the sclera the vision was found to be  $\frac{20}{200}$  and no detachment was seen.

ALLING.

LAWSON (114, **Detachment of the retina at the ora serrata**) reports a case of detachment of the retina at the ora serrata which followed as the result of a bomb which burst close by wounding the subject in several places but not in the eye.

T. HARRISON BUTLER.

Marcus Gunn in his classical paper upon the retinal signs of arteriosclerosis (*Trans. Ophth. Soc. U. K.*, vol. xviii.) laid down four cardinal signs:

(1) A tendency to *tortuosity of the arteries*, especially the small ones.

(2) *Variation in the caliber* of the vessels, especially sudden diminution for a short length.

(3) *Alteration in the normal light streak* which becomes much brighter and more sharply defined.

(4) *Indentation of the veins* by the hardened artery. Considerable indentation is followed by obstruction with all its sequelæ. These classical observations have been confirmed by hundreds of observers, and no additional sign of any importance has been discovered. De Schweinitz emphasizes the brick-red coloration of the disk as a late but important sign.

Gunn was one of the first to note that these signs were not confined to old age, and that old age was not the sole cause of the changes.

At first the prognosis was regarded as very bad, but the author some years ago came to the conclusion that this view must be modified. In following up the history of these patients he found that some with advanced mischief, even with retinal œdema and hemorrhages, improved marvelously, and was still more surprised to note that in some the cardinal signs disappeared.

This view is strikingly confirmed by a case which is cited. A patient of Dr. Crofton, aged 60, had general symptoms of arteriosclerosis. He was examined by Mr. Worth who discovered advanced signs of arteriosclerosis in the retinal arteries including the "silver wire" appearance. After three months' treatment Worth found that the retinal arteries were normal for a man of his years. BARDSLEY (110, **Retinal signs of arteriosclerosis**) has observed these changes occur with *startling rapidity* even in a day or two! He was forced to the conclusion that the classical Gunn signs do not *necessarily* denote arteriosclerosis. His researches upon the blood pressure of these patients has led him to the conclusion that high blood pressure alone can account for some of the classical signs. He can now distinguish with the ophthalmoscope the signs which belong to sclerosis and those proper to high pressure.

In simple high tension:

(1) The vessels have the appearance of *uniform* distention.

(2) The light streak is *broadened* out: it may be greatly expanded covering the whole breadth of the vessel.

(3) The light streak is much brighter than normal, the brilliancy increasing with the tension until at a very high record it becomes like bright copper wire, not silver wire.

(4) The tight arteries indent veins. The degree of indentation is proportional to the pressure.

The following signs indicate actual sclerosis:

(1) *Irregular* tortuosity, especially of the smaller twigs.

(2) Increased brilliancy of the light streak, which at the same time appears to be *narrower* and more central.

(3) Irregularity of caliber and beading are sure signs of sclerosis.

(4) General diminution in the size of the vessels and a "silver wire" appearance indicate advanced sclerosis.

Naturally the conditions of high tension and sclerosis often coincide but by no means always.

Conditions of acute toxæmia give the same ophthalmoscopic picture as that seen in cases with hypertension although the blood pressure is low.

T. HARRISON BUTLER.

The study of the disturbances of blood pressure in the retinal vessels has not passed even the threshold of speculation and theorizing, and, yet, its clinical significance is of vast importance. BAILLART (109, **An attempt to determine the arterial tension in the branches of the central retinal artery**) has tried to work out a practical method of measuring the blood pressure in the retinal blood-vessels by applying on the eyelid the sphygmomanometer of Bloch-Verdin, specially adapted for the eye by Cheron. A gradually increasing amount of pressure, measurable by the apparatus, is applied by an assistant on the eyeball while the observer watches the retinal arteries with the ophthalmoscope, using the indirect method. The moment the arteries begin to pulsate, the first reading on the sphygmomanometer is noted, which indicates the amount of pressure exerted on the eyeball that overcomes the diastolic pressure in the retinal vessels. Then, the pressure is increased until the pulsation ceases entirely, and again gradually released till the pulsation reappears. That moment a second reading of the sphygmomanometer is made, which



indicates the systolic pressure in the retinal vessels. Adding to each of these two readings the intraocular pressure, obtained beforehand with a tonometer, we obtain the systolic and diastolic blood pressure in the retinal blood-vessels. The author has examined by this method fifty healthy individuals between 25 and 47 years of age and found an average of 67mm diastolic and 98mm systolic pressure. In another series of younger individuals he found 61mm and 86mm respectively.

SCHOENBERG.

FRENKEL'S (112, **Circular folding of the retina produced by contusion**) patient, an infantry sergeant, was exposed to terrific explosions of shells in the trenches and, without having been hit, he noticed that his right eye was blind. He reported for medical examination about eight months later and the diagnosis was made of optic neuritis and chorioretinitis in the right eye. Wassermann positive. After a few months of anti-luetic treatment he came under the observation of the author who found a peculiar condition of the fundus, consisting of a partial optic atrophy and five white circles arranged above and below the papilla. Two such circles above the papilla, each the size of about 1mm showed a few small white dots along the arteries passing within the zone of these circles. Two of the lower circles are somewhat larger than the upper ones; one of them presented at its lower periphery a hemorrhage, partly absorbed. The retina adjoining it was whitish. Finally a fifth line, occupying three fourths of a circle, situated in the inner lower quadrant of the fundus, presented three whitish patches, probably due to old hemorrhages. The left eye was normal. The author regards this unusual condition as due to circular folds of the retina, produced by the impact of the posterior pole of the eyeball against the tip of the orbit during a sudden increase of air pressure, caused by the forceful explosions of shells at a very short distance away, about two meters.

SCHOENBERG.

Aneurysms of the retinal arteries, apart from those variations in caliber associated with arterial disease, are extremely rare. PRINGLE (115, **Multiple aneurysms of the retinal arteries**) reports the following case. Private C. J., aged 23, had noted defective vision for eighteen months. V. R.  $\frac{6}{5}$ ; V. L.  $\frac{6}{5}$ .

*Left Fundus.* On following the superior temporal vessels outwards to a distance of about three disk diameters a large leash of medullated nerve fibers begins to make its appearance.

Just below this leash in the course of the lower branch of the superior temporal artery a string of aneurysms is seen. These with the vessel give the appearance of a Higginson's syringe with multiple bulbs. The condition is clearly shown by a beautiful colored plate of the fundus. The disease is very rare, and no ophthalmologist should fail to examine this instructive drawing. Unfortunately no mention is made of a Wassermann test.

T. HARRISON BUTLER.

TAYLOR and FLEMMING (116, **Bilateral glioma of the retina with multiple metastases**) describe a case of a child aged three who died from glioma in each retina with metastases in the meninges, bones, mesenteric glands, ovary, and elsewhere. The metastatic growths had the characters of a small round-celled sarcoma. The primary growth in the retina showed no neuro-epithelial rosettes. Photographs of the excised eyes and of the microscopic sections are included in the paper.

T. HARRISON BUTLER.

GRIFFITH (113, **Hereditary glioma of the retina**) gives an account of two families in which glioma retinae in the mother was transmitted to several children.

*The Smith Family:* Mother, aged 22, had had the right eye removed for glioma, by Mules, when nine months old.

Mrs. Smith had six children four of whom developed double glioma.

*The Jones Family:* Mother had the right eye removed for glioma, by Glascott, when two and a half years old.

There were three children; two had unilateral glioma and one bilateral.

Griffith states that all the eyes showed typical glioma retinae. He states that he can only find records of six other examples of this tragic inheritance. In two the disease was transmitted from the father.

T. HARRISON BUTLER.

XX.—THE OPTIC NERVE AND VISUAL TRACT.

117. BOURGUET and RONNAUX. **Bilateral optic neuritis cured by a puncture of the corpus callosum.** *Annales d'oculistique*, cliii., 7.
118. DOR, L. **Retrobulbar neuritis.** *Clinique ophtalmologique*, viiii., 5.
119. JOCQS. **Retrobulbar neuritis and multiple sclerosis.** *Ibid.*, viiii., 5.
120. LENOIR, W. **Acute infectious retrobulbar neuritis; semeiology and complications.** *Annales d'oculistique*, cliv., 2.
121. MENACHO, M. **Congenital pigmentation of the optic nerve.** *Ibid.*, cliv., 5.
122. SOLLIER, P., and JOUSSET, X. **Nitro-phenol optic neuritis.** *Clinique ophtalmologique*, viiii., 2.

BOURGUET and RONNAUX (117, **Bilateral optic neuritis cured by a puncture of the corpus callosum**) report the following case. The patient, a war prisoner, 24 years of age, complained of headaches and loss of sight following an attack of gripe. His vision was O. D. = P. L. O. S.  $\frac{3}{30}$ . The ophthalmoscopic examination revealed a bilateral optic neuritis. No other neurological symptom. With the assumption that the gripe produced an obstruction of the foramen of Magendie through which the cerebrospinal fluid flows from the fourth ventricle into the subarachnoid space, followed by an increase of intracranial pressure and optic neuritis, the authors decided to establish an artificial communication between the lateral ventricles and the subarachnoid space. For this purpose they punctured the corpus callosum, under local anæsthesia, and obtained an almost immediate improvement. The vision returned to normal in a few weeks and the papillitis cleared up very rapidly.

SCHOENBERG.

DOR (118, **Retrobulbar neuritis**) is convinced that in nineteen times out of twenty the cause of retrobulbar neuritis lies in an inflammatory condition of the roots of the two upper bicusps and a periostitis surrounding them. The diseased teeth are on the same side as the affected eye. The simplest way to find out the tooth trouble is to press upon the gums in the region of the bicusps. Pressure elicits pain if there is a periostitis. He advises dentists always to extract diseased bicusps, never to fill them.

SCHOENBERG.

JOCQS (119, **Retrobulbar neuritis and multiple sclerosis**) publishes the history of a case who had a retrobulbar neuritis

of one eye and a large central scotoma. The condition cleared up entirely in about two months. Ten years later the patient developed multiple sclerosis.

SCHOENBERG.

Based on sixteen observations LENOIR (120, **The acute infectious retrobulbar neuritis**) summarizes the salient points of characterizing the acute infectious retrobulbar neuritis. Twelve of his patients gave no history of any infection preceding or accompanying the eye trouble, four had rhinitis. The vision was abolished entirely in three, reduced to perception of light in six, and below  $\frac{1}{20}$  in two. Improvement usually began at the end of the first week. The ophthalmoscopic changes depend on the seat of the inflammatory process, and the author classifies his cases in two types: *anterior* or *juxtabulbar*, in which the inflammation is very near the eyeball, and *posterior*, when the process is farther back, behind the point of entrance of the retinal blood-vessels.

In six cases there was an optic neuritis, in three papillitis, in two hyperæmia of the optic disk. The others showed no ophthalmoscopic changes. The changes in the visual field were: absolute central scotoma in ten, irregular contraction of the visual field in three, relative central scotoma in one. The usual course was benign. It cleared up entirely in from four to eight weeks. The author warns the reader to exclude multiple sclerosis, syphilis, and Leber's hereditary optic atrophy before a definite diagnosis is made of acute infectious retrobulbar neuritis.

SCHOENBERG.

SOLLIER and JOUSSET (122, **Nitro-phenol optic neuritis**) have seen fifteen workers from munition factories, who after handling nitro-phenol for several months began to complain of cloudy vision, cramps, and pricking sensation in the legs. The ophthalmological examination revealed the existence of a retrobulbar neuritis which usually ended in an optic atrophy. The authors believe that this neuritis is due to the toxic action of the nitro-phenol. The condition develops very slowly and a discontinuation of the work at an early period of the trouble is very important for its successful treatment. All the cases reported in this paper came under observation months after the visual trouble had begun.

SCHOENBERG.

MENACHO (121, **Congenital pigmentation of the optic nerve**) reports on a case of annular pigmentation of the optic disk. There was also a patch of thin sclera with the choroid showing through near the upper margin of the cornea. He believes that the coincidence of the pigmentation on the disk with the aplasia of the sclera speaks in favor of a disturbance of embryonic origin and assumes that the pigment had its origin from an inclusion of a portion of the pigment-epithelium layer of the retina.

SCHOENBERG.

# XXI.—ACCIDENTS, INJURIES, FOREIGN BODIES, PARASITES.

123. COSSE and DELORD. Right homonymous hemianopsia complicated by a left inferior quadrant bilateral hemianopsia. *Annales d'oculistique*, cliv., 2.

124. DOR, L. Intraocular foreign bodies. *Clinique ophthalmologique*, viii., 4.

125. EATON, E. M. The more accurate determination of the position of foreign bodies in their relations to the eyeball and its component structures. *British Journal of Ophthalmology*, December, 1917.

126. FRENKEL, H. Traumatic syndrome of the anterior segment of the eyeball. *Annales d'oculistique*, cliii., 6.

127. MILLER, G. V. Injury to the eye complicated with abscess of the brain terminating fatally; autopsy; two cases. *British Journal of Ophthalmology*, December, 1917.

128. MORAX, V. Remarks on the prognosis and technique of extraction of intraocular magnetic foreign bodies. *Annales d'oculistique*, cliii., 10.

129. MORAX, V. Multiple intracranial projectiles causing a variety of ocular syndromes: homonymous hemianopsia, neuromyolytic keratitis, paralysis of associated movement to the right. *Ibid.*, cliv., 5.

130. MORAX, V., and MOREAU, F. The ætiology of injuries of the eyes by projectiles of war. *Ibid.*, cliii., 8.

131. PETIT, P. Remote consequences of war injuries of the eye and of intraocular foreign bodies. *Ibid.*, cliii., 7.

132. PETIT, P. Two cases of burns of the eye with a caustic liquid. *Ibid.*, cliii., 11.

133. SCHNEIDER, E. R. Cases of tetanus following ocular injuries. *Ibid.*, cliii., 9.

134. SHUMWAY, E. A. Contusion of the eye with rupture extending across the optic nerve. *Ophthalmic Record*, April, 1917.

DOR (124, **Intraocular foreign bodies**) relates his results in twenty cases of foreign bodies in the vitreous treated during

twelve months: four enucleations; four remained with an atrophic eyeball; seven eyes conserved retaining their normal appearance, but with intraocular hemorrhages, susceptible of absorption; two cases remained with a traumatic cataract, of which one was successfully operated on, while no intervention was made in the other; three eyes were preserved with a vision above 1<sup>0</sup>.

SCHOENBERG.

EATON'S (125, **The more accurate determination of the position of foreign bodies in their relation to the eyeball and its component structures**) paper is highly geometrical and cannot well be abstracted without the reproduction of the numerous diagrams.

T. HARRISON BUTLER.

MORAX (128, **Prognosis and technique of extraction of intraocular magnetic foreign bodies**) states that of fifty-nine cases of foreign bodies in the vitreous thirteen ended in enucleations, eighteen remained with zero or very poor vision, twenty with useful vision, and eight recovered completely. Of fifteen cases with foreign bodies in the lens, iris, or anterior chamber, one eye had to be enucleated, one remained blind, four had satisfactory vision, and nine gave very good results. The prognosis was best in eyes with small foreign bodies, weighing from three to five milligrams. Regarding the preference some authors have for the giant over the small magnet, or vice-versa, Morax thinks that both are necessary, but that the small magnet is the most indispensable.

SCHOENBERG.

PETIT (131, **Remote consequences of war injuries of the eye and of intraocular foreign bodies**) advises that patients with such injuries should be carefully watched for a long time, even if the eye appears perfectly calm. He has seen about forty cases in which the X-ray or clinical examination did not reveal the presence of a foreign body in the eye. Later on when the enucleation became necessary, minute particles were found in the ciliary body, vitreous, etc.

SCHOENBERG.

MORAX and MOREAU (130, **The ætiology of injuries of the eyes by projectiles of war**) call attention to the fact that in

this war there has been a larger percentage of eye injuries than in any other war. Of 697 eye injuries, the most frequent (341) were due to fragments of shells; and the least frequent to bombs and rifles. The eyes are injured to a greater or less degree by direct or indirect contusion, rupture of the ocular tissues, infection, trauma or section of the optic nerve or of the oculomotor nerves, or the vision is impaired by intracranial lesions of the visual paths or centers. The amount of damage sustained by the ocular tissues depends very much on the size and velocity of the foreign body. They classify the foreign bodies according to their weight: large, 5 to 10 grams, medium, 0.30 to 5 grams, small, 0.001 to 0.25 grams. Of the 341 cases 75 were due to large, 96 to medium, and 170 to small-sized fragments of projectiles. The functional impairment resulting from the eye injuries in 697 cases was as follows: (1) 33 cases of trauma of the cranium resulting in 14 in a permanent homonymous hemianopsia; (2) 293 enucleations; (3) vision reduced to less than  $\frac{1}{20}$  in 194 cases; (4) vision reduced to  $\frac{1}{10}$  to  $\frac{1}{20}$  in 36 cases; (5) only 160 have recovered vision to a useful degree. Regarding the prevention of entrance of these foreign bodies into the visual organs, the authors are very much in favor of various protective spectacles devised for such a purpose since the beginning of the war.

SCHOENBERG.

MORAX (129, **Multiple intracranial projectiles causing a variety of ocular syndromes: homonymous hemianopsia, neuroparalytic keratitis, paralysis of associated movement to the right**) reports the history of a soldier, wounded in the right occipital region by a fragment of a shell, who developed a multiplicity of symptoms of unusual interest. The symptoms following the injury were during the first month: loss of memory, inability to stand erect or to walk, marked diminution of vision to movements of the hand at 50cm. Wound on the right side of the occipital region entirely healed. X-ray picture revealed the presence of two foreign bodies; one in the region of the tentorium cerebelli "on the median line" and one in the posterior portion of the right occipital lobe. During the second month of illness the vision became normal and the general condition improved, but a slight attack of paralysis of the right trigeminus and left facial nerve appeared with a

mild neuroparalytic keratitis on the same side. Left homonymous hemianopsia, fundus normal, inability to look to the right. Some disturbance of gait and tendency to turn around, cerebellar. About two and a half months after the injury the patient became suddenly prostrated, complained of slight headache, and died the same night. The autopsy revealed: (1) Subdural hæmatoma at a point corresponding to the scar of the scalp in the occipital region. (2) Purulent meningitis, mostly developed in the region of the cerebellum. (3) The presence of three foreign bodies. One, in the right occipital lobe, surrounded by a hemorrhagic mass. Another, after injuring very gravely the brain substance of the occipital lobe, was arrested under the tentorium cerebelli. The third, in the right cerebellar hemisphere, surrounded by a purulent mass. This was the starting point of the suppurative meningitis. The author explains the visual and motile disturbances in the following manner: (1) The left hemianopsia was due to the injury of the right occipital lobe. (2) The neuroparalytic keratitis was produced by the suppurative process in the right cerebellar lobe, extending on the right side of the pons at the point of emergence of the fifth cranial nerve. (3) The paralysis of associated movements to the right may be attributed to the cerebellar abscess or to the involvement of the nucleus of the right sixth nerve. The latter appears more probable to the author. It is worth noticing that there were no nystagmus, no papilloedema, fever, or any other symptoms pointing to a cerebellar abscess. Pulse and temperature remained normal throughout.

SCHOENBERG.

COSSE and DELORD (123, **Right homonymous hemianopsia complicated by a left inferior quadrant bilateral hemianopsia**) report the case of a soldier, who had been wounded in the occipital region by a fragment of a shell, and had a normal acuity of vision but behaved like a blind man on account of his field of vision. All that was left of the field was a quadrant in the upper left portion. The condition was stationary for three months, during which time he was under observation. No other neurological symptoms. The X-ray examination revealed the presence of a depression of the occiput in the median line, the seat of a trephining performed after the injury,



and two spicules of bone, one on each side, localizable, according to the method of Marie and Chatelin, above the visual centers at the tip of the occipital lobes. The injury of the brain tissue must have extended to the upper margin of both calcarine fissures, in order to give the extensive loss of the visual field. The case is almost unique in literature, one similar case reported by Sack being mentioned by Rochon-Duvigneaud in the French *Encyclopédie d'Ophthalmologie*.

SCHOENBERG.

FRENKEL (126, **Traumatic syndrome of the anterior segment of the eyeball**) describes under this title the following group of symptoms: (1) Superficial loss of substance of the skin around the orbit. (2) Small perforation at the limbus corneæ and small iridodialysis. (3) Subluxation of the lens. (4) Subcapsular opacity. (5) Normal fundus but vision markedly reduced from  $\frac{1}{20}$  to  $\frac{1}{50}$ . He reports twelve cases with this syndrome.

SCHOENBERG.

The rarity of such cases induced SCHNEIDER (133, **Tetanus following ocular injuries**) to publish the history of his own patient. A man 36 years of age was hit with a whip in the left eye which caused a perforating injury of the cornea with prolapse of iris. About three weeks after the accident he came to the hospital with panophthalmitis and beginning tetanus. He died three days later. He presented on his admittance in the hospital the characteristic symptoms of cephalic tetanus, difficulty in deglutition, trismus, and paralysis of the seventh nerve. The author reproduces from literature nineteen histories of cases of tetanus following injuries of the eyeball.

SCHOENBERG.

P. PETIT (132, **Burns of the eye with a caustic liquid**) reports the cases of two soldiers who were hit at the same time and probably by the same fragments of an enemy's shell. Both had severe superficial burns of the scalp, numerous small wounds of the lids, and deep perforating ulcerations of the cornea of one eye. Enucleation had to be performed in each case. The author thinks that the lesions were due to some caustic liquid, probably phosphoric acid.

SCHOENBERG.

MILLER (127, **Injury to the eye complicated with abscess of**

**the brain)** reports the following cases. A boy received a kick over the right eye during a game of football. There was a small laceration of the upper lid, the eye was uninjured. Three days later he was admitted to hospital. Eight hours later he died with cerebral symptoms. The autopsy showed a fracture of the osplanum of the ethmoid, and also of the cribriform plate. There was an abscess in the frontal lobes. The boy was 9 years old.

A girl aged 6 was struck upon the eye with a stick. There was a wound of the plica semilunaris. Four days later she had an attack similar to the first child with Cheyne Stokes breathing, from which she recovered. An incision was made next day, and pus evacuated from the orbit. Soon aphasia was noted and the left arm became paralyzed. The skull was trephined over the Rolandic area, but no pus was found. Eighteen days later the wound was opened up, and pus squirted out. A large hernia developed and the child died three months later.

A very extensive frontal abscess was found at the post-mortem examination.

T. HARRISON BUTLER.

SHUMWAY'S (134, **Contusion of the eye with rupture extending across the optic nerve**) patient was struck in the eye with a stone. There was hemorrhage into both aqueous and vitreous chambers and the fundus could not be seen until five months later when the blood and exudate had disappeared. A connective tissue band was seen running across the lower outer quadrant of the disk which partly filled in a deep depression at the border of the nerve head. The blood-vessels were interrupted as they crossed this area and were much reduced in caliber beyond the band. The nerve was atrophic and the eye blind. The injury was evidently a tear across the nerve and separation of the tissues at the level of its entrance into the eyeball. Several cases of partial and complete avulsion of the optic nerve have been reported.

ALLING.



ILLUSTRATING DR. POSEY'S "REPORT OF FOUR CASES OF ORBITAL TUMORS SUCCESSFULLY  
REMOVED WITH PRESERVATION OF VISION THROUGH AN ORBITAL INCISION ALONG  
THE EXTERNAL ORBITAL RIM."



Before operation.



After operation.



Before operation.



After operation.

## ARCHIVES OF OPHTHALMOLOGY.

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### REPORT OF FOUR CASES OF ORBITAL TUMORS SUCCESSFULLY REMOVED WITH PRESERVA- TION OF VISION THROUGH AN ORBITAL IN- CISION ALONG THE EXTERNAL ORBITAL RIM.\*

BY DR. WM. CAMPBELL POSEY, PHILADELPHIA, PA.

(With four illustrations on Text-Plate XVII.)

CASE I.—Male, 48 years of age. A continuous and gradual proptosis of the right eye past six years. No pain in globe, but occasional temporal, supraorbital, and frontal headache. No history of trauma or inflammation. Health good. Wassermann negative.

*Examination.*—Right eye proptosed forwards 14mm in advance of its fellow, with downward displacement of about 5mm. The globe gives the impression of being about to pop out of the orbit. No irregularity of orbital rim, no palpable mass. No pulsation. Globe cannot be pushed back into orbit. Slight bulbar injection; anterior segment of globe otherwise negative. Pupil, 3mm, reacts to light and on accommodation. Moderate neuroretinitis. Left eye negative. V. R. E. =  $\frac{5}{6}$ ; L. E. =  $\frac{5}{6}$ . X-ray examination of orbit and surrounding sinuses negative. Nasal examination and transillumination of the sinuses negative.

*Operation.*—Ether. Incision over outer rim of orbit, as directed by Krönlein. After dissection of the tissues, a large encapsulated tumor, much the shape and size of the eyeball, found posterior to the eye and superior to the optic nerve, but without connection with either structure. Mass removed in

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\* Read at meeting of the American Ophthalmological Society, New London, July, 1918.

toto through the incision which was extended superiorly to give room for the exit of the tumor mass. Healing prompt, although for some days there was marked proptosis of the eyeball forwards in consequence of the hemorrhage occasioned by the trauma of the operation. Two months later, proptosis reduced to 4mm. Some ptosis and restriction of the outward movements of the eye. Both of these conditions are, however, rapidly subsiding. Vision as before operation. Some subsidence in the neuroretinitis.

*Pathological Report.*—Macroscopically the specimen consisted of a hard oval-shaped mass measuring 22 x 35mm in its longest diameter. The external surface was smooth. On section it was encapsulated. The tissue was white in color, resembling smooth muscle. Microscopically the tumor was encapsulated, the capsule consisting of encircling layers of long spindle cells arranged compactly in a manner resembling smooth muscle. The interior of the tumor was composed of the same type of tissue, less compactly arranged with a tendency to the formation of whirls. The blood-vessels were typical, and in most part were in the center of the whirls above mentioned. Diagnosis, spindle cell sarcoma.

CASE 2.—Male, aged 29 years. Gradual protrusion of right eye past five years. No trauma. No inflammatory symptoms.

*Examination.*—Right eye proptosed directly forwards 10mm in advance of its fellow. Health good. No traumatism. No pulsation. Impossible to press globe back into orbit. No orbital mass palpable. Globe negative. X-ray and nasal examination of orbit and accessory sinuses negative. Wassermann negative. V. R. E. =  $\frac{5}{7.5}$ ; L. E. =  $\frac{5}{8}$ .

*Operation.*—Ether. Incision as for Krönlein procedure. Large encapsulated tumor resting upon optic nerve below found immediately behind globe without connection with adjoining structure. Readily removed by blunt dissection. Healing prompt. At end of three months, proptosis entirely gone; ocular movements free in all directions. Scar over wound almost invisible.

*Pathological Examination.*—Macroscopically the specimen consisted of a round mass measuring 24mm in diameter. It was spongy to the touch. The center consisted of a network of fibrous tissue. Microscopically the specimen was composed throughout of dense fibrous tissue, most of which was arranged in a crinkly manner, and was devoid of nuclei. The arrangement was loose, presenting many open spaces, which were lined in some instances by a very thin endothelium. The growth was spongy, the sponginess being

attributed to numerous open spaces within the aforementioned but loosely arranged fibrous tissue. Diagnosis, fibroma.

CASE 3.—Female, aged 27 years. Right eye prominent since early childhood, but no apparent increase in past ten years. Health good. Wassermann negative.

*Examination.*—Right eye proptosed 8mm in advance of its fellow. Smooth hard mass readily palpated at upper, outer angle of orbit, extending downward as far as outer commissure. V. R. E. =  $\frac{5}{8}$ ; L. E. =  $\frac{5}{8}$ . Incision made over mass at outer angle of orbit. Cyst with firm capsule and dermoid contents removed without difficulty. Recovery rapid. At end of month proptosis almost entirely disappeared, some ptosis and limitation of external motion of eye.

*Pathological Examination.*—Dermoid cyst.

CASE 4.—Female, aged 18 years. Gradual protrusion of left eye, without inflammatory symptoms, for past eight months, this condition being attributable to an attack of typhoid fever three years previously, as a sequel of which the postcervical and submaxillary glands of the left side remained permanently swollen. One gland ruptured spontaneously. General health good. Family history negative for tuberculosis. Has never had diplopia, but covers left eye when reading to avoid seeing same line twice.

*Examination.*—Left eye proptosed down and somewhat out, 7mm in advance of its fellow. Under the upper lid at the outer third a firm movable mass, somewhat tender on firm pressure, is readily palpable, which gives the impression of being slightly lobulated and of a glandular nature. V. R. E. =  $\frac{6}{8}$ ; L. E. =  $\frac{6}{8}$ . The posterior border of left sternomastoid muscle is lined with glands varying in size from a pin to a small hickory nut. Similar glands are situated in the submaxillary region. Some suggestion of enlargement of middle lobe of thyroid. Von Pirquet and Wassermann negative. Moderate anæmia. X-ray and nasal examination negative.

*Operation.*—Ether. Incision made along and below left supraorbital ridge at the outer part, immediately over the palpable mass. Tumor the size of a large almond and surrounded by a firm capsule exposed and removed by blunt dissection, permitting eyeball to recede into its normal position. Healing prompt. At end of a month, all proptosis had disappeared and the movements of the eye were normal.

*Pathological Examination.*—Macroscopically the speci-

men consisted of a hard oval-shaped mass measuring 28 x 15 mm in its longest diameter. It was encapsulated. On section, tissue resembled that of a gland, the center of which was hard and fibrous. Microscopically the growth was encapsulated, the capsule contributing to the formation of trabeculae, which permeated throughout. The structure was that of an adenoma, the acini of which were small, each acinus being surrounded by a very definite connective tissue framework. The acini were about 70 microns in diameter. Near the capsule the acini arrangement was lost, probably due to compression in this region by the firm capsule. Diagnosis, adenoma.

With the exception of the fourth case, where the tumor was palpable and where the incision was naturally made directly over the mass, the precise location and the character of the neoplasm occasioning the proptosis were in doubt. The degree of the proptosis present suggested a tumor of considerable size in each instance, and the character of the displacement seemed to locate the growth in the posterior portion of the orbit. Under such circumstances it seemed advisable to enter the orbit through its outer part and to place the incision in such a way that if found desirable the outer wall of the orbit might be resected after the manner of Krönlein, thereby affording easy access to the orbital apex and permitting the easier removal of a growth from that region. Fortunately, however, in none of these cases was this found necessary. The author has performed the Krönlein procedure upon six cases, the notes of several of these having been submitted to this Society at previous meetings. All of these cases terminated successfully, and in only one was there any appreciable scarring, namely, in that of a child five years of age with a large dermoid cyst of the orbit, where the detached fragment of bone broke down, leaving for a time a discharging fistula.

All those who have performed the Krönlein operation, however, confess to the difficulty they encountered in displacing the wedge-shaped fragment of bone, resected from the outer angle of the orbit, in toto. On account of the contour of the orbital ridge and the contents of the orbit, it is impossible to employ straight saws with advantage. When chisels are employed, the bone is apt to split and it is practically impossible to remove the apex of the wedge as recommended by



the author of the operation. In several of his cases the writer has first drilled the bone with a dental engine and then employed the chisel, but even with this modification the apex of the triangle of bone has remained. Some years ago he was much impressed by an article by Magitot and Landrieu, of Paris, translated by Ziegler, of Philadelphia, which contained what appeared to be a valuable modification of the Krönlein method. As the description of this method may have escaped the attention of some of the members of this Society, it may not be amiss to quote the method in extenso. As the authors follow the original Krönlein method precisely until the resection of the bone is reached, it will only be necessary to detail their observations from that point. They are as follows:

Now, we proceed to the bony sections. This is without doubt the most delicate stage. The reason for this is on account of the extreme hardness of the orbital border. The bony tissue of this region is certainly the most resistant of the body and its eburnation in aged subjects is normal. It is not necessary to depend upon chisels in order to finish the operation. Fortunately, we have saws. But straight saws are inconvenient. Using them horizontally, they no sooner enter the bony tissue than they injure the soft parts: for on one side are the muscles of the skin and on the other side are the eyeball and the eyelids.

In order that the bony block shall reach as deep as the pterygomaxillary fissure, it is necessary that the first section shall start from this fissure, passing from the depths to the surface. Therefore, every method which commences by cutting into the orbital border only succeeds in detaching the edge of the orbit, that is to say, only a small part of the external wall.

The orbital periosteum having been detached as far as the pterygomaxillary fissure, a malleable retractor is introduced into the cavity, pressing back the periosteum and the eyeball toward the nasal side. The bony border having been freely exposed by a separator (rugine), the temporal aponeurosis is incised. The object is to reach the pterygomaxillary fissure and introduce into this orifice a wire saw which should emerge through the orbit. Therefore, in order to do this, one should open up the temporal fossa a little in order to permit the passage of a probe or bodkin. With the aid of a grooved director one should then detach the whole exterior surface of the orbital apophysis of the

malar. Above all, the operator should bear in mind that it is in the angle formed by the malar apophysis and the zygoma (a great deal lower than is generally believed) that the probe must be introduced in order to penetrate into the orbit through the pterygomaxillary fissure. It is important at this point to press backward the muscular mass. It should be held back tightly by a retractor entrusted to an assistant.

Of course, one should avoid detaching the exterior periosteum of the temporo-malar, because it is through this membrane that the bony flap, dislocated and put back in place, is nourished. Then, taking a flexible bodkin, or if it is necessary, a grooved silver cannula, or better, a malleable conductor, the surgeon should give it a somewhat curved form and fasten to it the wire saw of Gigli. It is necessary then to recognize the location of the pterygomaxillary fissure. This fissure, concealed by fatty tissue, should be easy to locate with the end of a blunt instrument. It is well to note that the operator always has a tendency to look too high for it. It should be borne in mind that it is to be found at about 2.5cm from the orbital border formed by the *body* of the malar.

The pterygomaxillary fissure being found, the operator should introduce into it the flexible bodkin until he sees the point of the instrument emerge into the orbit, seizing it with a hemostatic forceps and drawing it toward him. He should then attach and introduce into the fissure the wire saw which he then disengages from the conductor.

There is nothing more to do now but to resect the orbital wall. The incision should be made sufficiently low in order to cut off the base of the orbital apophysis of the malar. The section, being easy, is made in a few seconds by the drill-bow movements imparted to the saw, which the two hands of the operator must hold *tightly stretched* in order to avoid kinks or breaking.

The section being finished, the surgeon searches at the extremity of the orbital apophysis of the malar for *the suture which unites the malar to the temporal*, the location being quite visible. Having recognized it he marks it with a protected saw, or in its absence, a very small straight saw, but the depth of this groove should not exceed 5 millimeters. The saw is then laid aside and the operator takes a small graver-chisel which he holds vertically, placing the cutting edge of it in the groove made by the saw. All that is necessary now is a simple blow of the mallet administered very moderately on the chisel in order to detach the *whole orbital apophysis of the malar*, as far as the pterygomaxillary fissure.

The bony block being liberated without splintering is

then *displaced* without difficulty and ultimately *put back in place* at the end of the operation.

Although the writer has practiced this method upon the cadaver, he regrets that the opportunity has not arisen to perform it upon the living subject. Although a number of cases of orbital tumor have presented themselves, the growths were removed in all with the preservation of the globe without the necessity of resecting the bone; indeed, it would appear from the author's experience that this step is necessary only in cases of tumor of the optic nerve, or in tumors situated at the apex of the orbit, which are of such magnitude that they cannot be forced between the globe and the external wall of the orbit, without injury to the former. While the modification of the Krönlein method suggested by these authors seems rational and feasible, the author confesses to having encountered great difficulty in inserting the Gigli saw through the pterygomaxillary fissure into the orbit. It is not easy to deflect the temporal muscle sufficiently to expose the fissure, and the angle at which the aneurysm needle must be made to enter the fissure is an awkward one. It is possible, however, to follow the steps outlined by the authors on the cadaver, and it may be easier, as oftentimes happens in other operative procedures, to perform it upon the living subject. The writer hopes that this method will be given a trial by others.

MICROSCOPIC FINDINGS IN A CASE OF CORALLIFORM CATARACT, WITH REMARKS ON CONGENITAL CATARACTS IN GENERAL.<sup>1</sup>

BY DR. F. H. VERHOEFF, BOSTON, MASS.

(From the Massachusetts Charitable Eye and Ear Infirmary.)

(*With two illustrations on Text-Plate XVIII.*)

CORALLIFORM cataract was first described as such by Marcus Gunn (1) in a brief communication to the Ophthalmological Society of the United Kingdom in 1895. His description of the clinical appearance of the cataract was as follows: "On focal illumination, rounded and oblong gray and white opacities are seen grouped towards the center of the lens, so as to resemble a piece of coral growing forwards and outwards from this point. The oblong opacities represent somewhat conical tubes, and their anterior extremities consequently appear as opaque circles or ovals. There are numerous very fine iridescent crystals throughout the clear part of the lens."

Since Gunn's report, cases of coralliform cataract have been described by Nettleship (2), Fisher (3), Langenhans (4), Kipp (5), Harman (6), and Stieren (7). Langenhans was apparently unacquainted with Gunn's case, but from his description it seems certain that his case was similar. Judging by the histories of the cases the condition is certainly present early in life, and is presumably congenital, although thus far it has not actually been observed at birth. It seems to remain almost if not quite stationary. Nettleship has shown very definitely

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<sup>1</sup> Read at meeting of the American Ophthalmological Society, New London, July, 1918.

ILLUSTRATING DR. VERHOEFF'S ARTICLE ON "MICROSCOPIC FINDINGS IN A CASE OF CORALLIFORM CATARACT, WITH REMARKS ON CONGENITAL CATARACTS IN GENERAL."

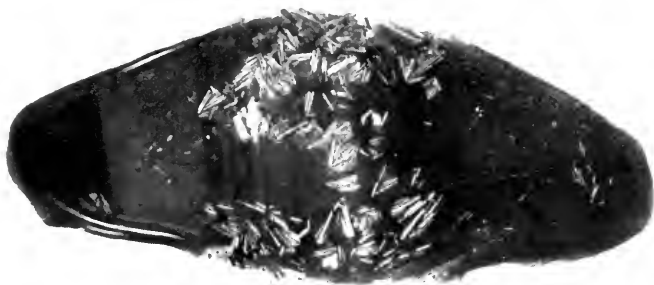


FIG. 1.—Coralliform cataract, showing arrangement of protein crystals in lens. Hematoxylin and eosin. Photo x 11.



FIG. 2. Protein crystals in lens. Note that degenerative changes in the lens substance occur only in relation with large masses of crystals. Hematoxylin and eosin. Photo x 50.



that coralliform cataract is hereditary. He relates the case of the Betts family, to which Gunn's patient also belonged, in which thirty persons in four generations were known to have been affected. Two children of this family had lamellar cataract, whereas their fathers had the coralliform variety. Nettleship remarks that there was nothing special in the health of the Betts family. The brother of Fisher's patient had congenital cataract, and Harman reported four cases of "axial" or coralliform cataract as occurring in three generations of one family.

So far as I can ascertain, the case I have to report is the only one in which a coralliform cataract has been examined microscopically.

#### CASE.

Arthur B., aged 39 years, was admitted to the Massachusetts Charitable Eye and Ear Infirmary, August 9, 1912. The patient states that he first became aware at the age of ten that his sight was imperfect and that it has grown no worse since then. The visual acuity of the right eye is  $\frac{5}{200}$ , that of the left,  $\frac{2}{200}$ , and in neither eye is improved by glasses. On examination by oblique daylight illumination each lens shows the same picture. Ramnifying out from the posterior cortex is an irregular brilliantly white structure closely resembling a miniature coral. Glistening particles, supposably cholesterine crystals, are also seen here and there in the lens. The lens substance is otherwise clear and permits a fundus reflex to be seen around the opacities.

On August 13, 1912, I removed the cataract from the right eye. The usual cataract incision was made with a Graefe knife, an iridectomy done, a large piece of the anterior capsule removed with toothed capsule forceps, and the lens expressed without difficulty. Almost no cortical matter remained behind, and healing was uneventful. On August 27th, I operated on the left eye in the same manner and the patient was discharged from the hospital, September 7th. On October 4, 1912, his visual acuity in each eye with sph. + 12  $\ominus$  cyl. + 1.50 ax. 180° =  $\frac{2}{200}$ .

#### PATHOLOGICAL EXAMINATION.

Both lenses were fixed in 10% formalin. The left was retained as a macroscopic specimen, the other was embedded in celloidin and anteroposterior sections made through its

center. Each lens measured 7 by 3mm in size, only a small amount of the cortex evidently having been lost at operation.

Microscopic examination of the right lens reveals a remarkable picture. The lens in general is normal, but embedded in it are numerous crystals of various sizes, some of them massed together, others occurring isolated. At the posterior and anterior poles the largest masses as well as the largest crystals occur, while towards the periphery the crystals are always small in size and become more and more isolated. The crystals are confined to an area 5.5mm in lateral diameter, and, with rare exceptions, only in the central portion of the lens are they found at the anteroposterior limits of the specimen. They show little tendency to follow the direction of the lens fibers, often lying at right angles to the latter. Immediately around each of the larger masses of crystals the lens substance has undergone marked cataractous changes, showing vacuolization and fragmentation. Otherwise the lens substance is normal, except that here and there it shows peculiar round or oval spots, .01mm to .04mm in diameter. The central portion of each of these spots stains in hematoxylin somewhat more strongly than the lens substance while its periphery stains faintly in eosin, so that the appearance of a ring is produced. These spots occur only near the periphery of the lens and never in close approximation to the crystals or to the cataractous changes around the latter.

When favorably situated in the sections the crystals are seen to consist of rectangular or rhombic plates. The largest seen measures .44mm in length. The thinnest of the largest crystals as seen in the sections is .01mm thick, so that it is probable that this is a close approximation to their actual thickness. The smallest isolated crystals are .08mm long and .004mm thick. Generally the surface of the crystals are parallel, but occasionally one edge is twice as thick as the other. Very rarely a crystal in the shape of a hexagon is seen. This may be the result of an oblique section of a rhombic plate. The crystals injured the edge of the microtome knife only slightly, so that it was possible to obtain sections 5-8  $\mu$  thick. Numerous cross-lines, however, can be seen on the crystals, produced by the passage of the knife. Scrapings made from the cut surface of the lens also showed the crystals to consist of rhombic plates.

In sections stained in hematoxylin and eosin by the usual method, the crystals remain colorless. If, however, the sections are placed in a .2% solution of water soluble eosin in 80% alcohol for twenty-four hours they take a strong eosin stain. The crystals stain most beautifully and intensely, however, in carbol-fuchsin. By staining sections



twenty-four hours in carbol-fuchsin and then differentiating in alcohol and acid alcohol, and finally treating them with a solution of permanganate of potassium followed by oxalic acid, the crystals are stained intensely red on an almost colorless background. The crystals are also stained by Lugol's solution of iodine. They fail to stain by Gram's method. They also fail to stain in phosphotungstic acid hematoxylin. When placed in a 1% solution of osmic acid for forty-eight hours, the lens substance and the crystals are stained the same color, a light brown.

The crystals are found to be insoluble in the cold, in concentrated hydrochloric acid, sulphuric acid, acetic acid, aqua regia, ammonium hydrate, sodium hydrate, acetone, chloroform, alcohol, ether, xylol, or oil of origanum. The crystals are not melted or affected in any way when sections are boiled in water or absolute alcohol. They are destroyed when sections are placed in a saturated, aqueous solution of potassium permanganate for two hours and then in a saturated solution of oxalic acid for a few minutes. When sections are placed in concentrated sulphuric acid on a slide, and sufficient heat applied to dissolve the lens substance, the crystals are also dissolved. When a section is dried on a slide and then heated over a flame, the crystals are not destroyed until the lens substance is charred and partially burned. When sections are placed in an aqueous alkaline solution of trypsin in an incubator for more than a week, the crystals fail to dissolve. When placed in a solution of pepsin in .4% hydrochloric acid they are dissolved completely in forty-eight hours, while the lens protoplasm is only partially digested. When placed in Millon's reagent for ten minutes to twenty-four hours, the crystals are stained an intense rose color, while the protoplasm of the lens substance itself is stained much less strongly. In other words, the crystals give a strong reaction for protein by this test.

Examination of the left lens which was retained as a macroscopic specimen also reveals the presence of crystals. This lens was carried through alcohol and xylol in an unsuccessful attempt to make it more transparent. Finally the xylol was removed and the specimen placed in glycerine. When the lens is viewed under the low power of the microscope, the opacities can be seen to contain numerous transparent rhombic slates.

It is evident that the lenticular opacities in this case were due almost wholly to the presence of crystals. Compared to the crystals, degenerative changes in the lens substance were

inconspicuous, occurring almost exclusively around the large masses of crystals, and being absent about the isolated single crystals. From this it seems certain that the crystals were primarily deposited in the clear lens substance and that the degenerative changes probably resulted from their presence. The round spots described as occurring in small number at the periphery of the lens differ in appearance from any lens changes I have previously seen. They have no resemblance to the vacuoles of congenital cataracts, and never occur in close approximation to the crystals. They somewhat resemble, however, the early degenerative changes of senile cataracts, and are therefore probably of the same origin as the latter.

Owing to the arrangement of the opacities in his case, Nettleship assumed that coralliform cataract was identical in nature with the variety of congenital cataract known as spindle, axial, or fusiform cataract, and this view has been accepted by subsequent writers. In my case, similarity to spindle cataract was not noted clinically, but examination of the hardened specimens and the sections shows some indication of a spindle arrangement. Thus, the crystals are most abundant in the axis of the lens and especially at its poles, while peripherally they follow more or less closely the outline of an expanded spindle. Nevertheless, I am convinced that coralliform is entirely distinct from spindle cataract. In the first place, its coral-like appearance is so striking that the appropriateness of the term coralliform is immediately obvious. In the second place, the two microscopic examinations that have been made of spindle cataracts in rabbits by Bach and Hess have shown changes that bear no resemblance to those found in my case. Both Bach and Hess found the opacities in spindle cataract to consist of degenerated lens fibers, and no mention was made of the presence of crystals. From his examination, Hess concluded that spindle cataract was due to delayed closure of the lens sac in the embryo, whereas in my case there was no indication of any malformation of the lens.

The exact nature of the crystals is not at once obvious. Their insolubility in ether, etc., excludes the possibility of their being cholesterine crystals, although in shape they are similar to the latter. Their failure to stain in phosphotungstic acid hematoxylin excludes hemoglobin or hematoïdin. Their

general insolubility and the fact that they stain in carbol-fuchsin by the tubercle bacilli method exclude all inorganic crystals. It occurred to me, therefore, that they might consist of the salt of some fatty acid, as calcium stearate. By adding calcium chloride to an alcoholic solution of stearic acid, rectangular crystals were obtained which had the same insolubility as those of the lens crystals. I was unable to stain them, however, and the possibility of the lens crystals being fatty acid in nature was of course completely excluded when I found that they gave the Millon reaction and could be digested by pepsin. That they could not be digested by trypsin is explained by their having been fixed in formalin.

Dr. E. W. Clap suggested to me that the crystals might be protein in nature and similar to protein crystals occurring in plants. It was largely due to this suggestion that the Millon test for protein was applied and found positive. Previously I had presented the problem to several physiological chemists who were unable to throw any light on the matter. Professor Osterhout suggested to me that the crystals were simply crystallized lens protein, and this suggestion seems to me certainly correct, since it explains their insolubility (after fixation in formalin), their staining reactions,<sup>1</sup> their positive response to the Millon test, and their digestibility in pepsin. So far as I know, this is the first instance in which protein crystals have been demonstrated in animal tissue of any kind.

The question arises as to the condition under which the crystals become deposited in coralliform cataracts. As already mentioned, Nettleship found two individuals with lamellar cataracts whose fathers had coralliform cataracts. This indicates a close relation between the two forms of cataract. Now it is well known that lamellar cataract occurs most often in children who show signs of rickets, so that it is fair to assume that this form of cataract is dependent upon a faulty calcium metabolism.<sup>2</sup> It seems more than probable, therefore, that

<sup>1</sup>I find that the protein crystals of a Brazil nut also stain differentially in carbol-fuchsin by the method for tubercle bacilli.

<sup>2</sup>Lamellar cataract is also sometimes associated with tetany, in which there is believed to be faulty calcium metabolism dependent upon parathyroid insufficiency. Tetany occasionally occurs in rachitic infants but it is not certain that both conditions are dependent upon the same constitutional factors.

coralliform cataract and lamellar cataract are both dependent directly or indirectly upon a faulty calcium metabolism. It is interesting to note, however, that in only one case of coralliform cataract reported, that of Langenhans, were there signs of rickets. This would tend to indicate some difference in the metabolic disturbances in the two conditions. Microscopic examinations of lamellar cataracts show that the opacities are due to the presence of fine vacuoles. It is evident that neither lamellar or coralliform cataract is dependent upon degeneration changes, as has been assumed, since in early cases such changes are absent about the vacuoles and crystals. Just what the vacuoles of lamellar cataract contain in the living lens has not hitherto been definitely determined so far as I can find, but the appearances that they present in microscopic sections indicate with considerable certainty that at some period they contained a fatty substance that has dissolved out. In this connection the following quotation from Stricker's monograph (8) seems to be of considerable significance:

"That there is a chemical difference between the inner and the outer portions of the lens seems to be attested by a fact which, though known for a long time and frequently discussed, has been variously accounted for; namely, that in the lens of all vertebrates and young animals which during life have been perfectly transparent, immediately after death, as soon as the animal becomes cold, the inner portion of the lens becomes cloudy. Michel drew attention to the fact and showed that the cloudiness in the center of the lens of cats, pigs, and calves would disappear if they were warmed up to 15 to 20 degrees C. This could be repeated as often as desired, and each time the lens would clear up again. This cloudiness is produced by the presence of innumerable roundish, highly refracting droplets in the central portions of the lens, and is not in any way connected with the decomposition of the contents of the lens, such as is produced by freezing. In these experiments there can be no question as to the separation of the water from the albumin. These droplets become less numerous as we go from within outward. Treated with alcohol and ether, their number becomes less, and they are reduced in size. Hence it appears that they must consist of a fatty substance, which has a very low melting point, though it is impossible to state anything more definite at this time. The interest which the appearance of these bodies at a low temperature arouses is

due less to their presence than to the fact that they occur in the inner lamellæ of the lens, that their number varies in different species of animals, and that their deposit ceases a few months after birth. From this it follows that in the chemical formation of the lens, especially in the older layers of the same, very pronounced changes take place. Since, according to Kuhne and Laptschinsky, the amount of fat is greater in the old than in the young, hence it cannot be due to a quantitative but rather a qualitative change."

These observations may explain why lamellar cataract is formed only in childhood or before birth. Their possible significance in this regard, curiously enough, seems hitherto to have been entirely overlooked. In the hope of throwing further light on the matter, I recently placed the lens removed from a 3½-weeks-old human infant, which had been fixed in formalin, in 1% osmic acid for five days. The lens was first bisected to ensure penetration of the solution. On microscopic examination I found a peripheral zone, thickly studded with blackened droplets. These droplets were readily bleached when treated with a saturated solution of potassium permanganate followed by oxalic acid, thus proving that they were not ordinary fat but that they were probably related to myelin.<sup>1</sup> If these findings are confirmed by further observations that I hope to make, they will prove conclusively that lamellar cataract is due to the undue persistence of lipoid droplets normally present in foetal lenses. Their persistence in cases of congenital cataract possibly could be explained by a faulty fat metabolism either dependent on or associated with the faulty calcium metabolism. Ultimately it is possible that they may be replaced by lime salts or other substances.

To explain the probable relation of coralliform cataract to faulty calcium metabolism seems difficult. Professor Osterhout informs me that the conditions under which the proteins crystallize in living tissues are not definitely known, but that almost any protein may be made to crystallize by withdrawing the salts from the solution in which it is dissolved. The possibility suggests itself, therefore, that the crystallization of the

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<sup>1</sup> Deutschman (9), who made the first microscopic examination of a lamellar cataract, assumed, but without proof, that the droplets described by him were of myelin.

lens protein in coralliform cataract may be dependent upon a deficiency of calcium or other salts in the blood or aqueous humor.

As to the time when the crystals are deposited, it would seem that owing to the softer consistency of the foetal lens the conditions are more suitable for their deposition in foetal life than after birth. The great abundance and large size of the crystals seem to exclude the possibility that they are precipitated in the fluid contents of the embryonic lens cavity.

Whatever the exact natures of the droplets in lamellar cataract and of the crystals in coralliform cataract may be, it seems sufficiently clear that the lens opacities in each case result from some metabolic disturbance and are not primarily due to degenerative changes in the lens itself. We may, therefore, classify congenital cataracts into two chief types, namely, developmental cataracts, exemplified especially by posterior polar cataracts, and metabolic cataracts, exemplified by lamellar and coralliform cataracts.

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## A CASE OF EVULSION OF THE OPTIC NERVE.<sup>1</sup>

By EUGENE M. BLAKE, CAPTAIN, M.R.C., NEW HAVEN, CONN.

IF one may judge by the paucity of references in the literature, evulsion of the optic nerve is a very rare condition. Undoubtedly the nerve is separated from the globe oftener than we realize, but because of other changes which make it impossible to examine the fundus and because the injury which produces the evulsion may be associated with grave cerebral symptoms and fatal termination, we are not permitted a great familiarity with this form of ocular traumatism. Natanson, writing in 1912, stated that his case was the sixteenth in the literature and since that time there are but two more cases, so far as I can find. The only complete description of evulsion occurs in the section of Injuries to the Optic Nerve in the *Graefe-Saemisch Handbuch*, with brief discussion in the *American Encyclopedia of Ophthalmology* and in Parsons's *Pathology of the Eye*.

The terms avulsion and evulsion appear to be used in the same sense by different authors and it would be well to employ one term consistently, rather than use two for the same condition. Avulsion is derived from the Latin *avellere*, meaning to pluck off, while evulsion comes from *vellere*, to pluck, and (*e*)—a plucking out. The latter word would seem to be the more descriptive of the two for that is what actually occurs. The optic nerve is plucked out of the globe at the point where it enters. Salzmann, in 1903, was the first to label the condition and he called it *evulsio optici nervi*.

The case which was seen by the writer occurred in a Russian Jew, aged 29, on May 14, 1917. Early in the morn-

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<sup>1</sup> Read at meeting of the American Ophthalmological Society, New London, July, 1918.

ing on that date, while driving a baker's wagon, he collided with a milk team. His own wagon was overturned, throwing him to the ground in such a manner that his head struck upon a trolley track, his wagon falling on top of him. He was brought into the New Haven Hospital at 7.30 A.M. the day of the accident and the following notes recorded upon his chart.

*Examination of Head and Neck.*—Calvarium. Scalp wound over left parietal region near mid-line about two inches long. Closed by black silk sutures. No line of fracture or depression felt in skull. Left eye badly lacerated and bulging from orbit—enucleated in the accident room. Right eye, both lids swollen and ecchymotic. Marked conjunctival hemorrhage. Pupil slightly dilated and does not react to light, nor has patient perception of light.

Ears—negative, no bleeding.

Nose—compressed and deviated to left side. Loose from its deeper attachments. Can insert fingers into antra.

Mouth—complete fracture of maxillæ, which are loose from their attachments. Both maxillæ apparently crushed upwards and backwards. No fracture of mandible detected.

Face—wound left side chin one inch long. Wounds of mucous membrane, inner side cheeks, especially on the left side, near angle of mouth.

Chest—fracture of left clavicle about center, dislocated anteriorly. Not much over-riding.

*X-Ray Report.*—Fracture of both maxillæ. Possible fracture right mandible. Fracture into left orbit. Multiple fractures of maxillæ, nasal and orbital bones. Marked displacement of fragments. Possible fracture of coracoid process. Fracture left clavicle and third rib.

The physical examination upon entrance into the hospital is given in detail as it affords an explanation of the mechanism of production of the evulsion of the optic nerve in this case. The patient was thrown from his wagon, falling upon his head in such a way that the force of the blow was directed upward and to the left. This accounts for the fracture and separation of the superior maxillæ and nasal bones and the extrusion of the left eye from the orbit, as evidenced by the location of the wounds. Evidently the direct line of force was upward and to the left, but enough pressure was disseminated to the right to produce just sufficient protrusion of the right eye to separate the optic nerve from the globe without rupturing the sclera and causing collapse of the eye, as occurred on the left side. It is as though one pulled an apple from its stem.

The eye ground was examined for the first time one week after the accident. The pupil was moderately dilated and



absolutely immobile. The media were perfectly clear and the anterior chamber was of normal, or only slightly increased depth. The region of the papilla was occupied by an excavation, the depth of which was at least 8 D. but was very difficult to estimate, as there were no details to be made out at the bottom of the excavation. In color it was nearly uniformly gray, and one had the sensation of looking into a deep well.

The diameter of the hole was slightly greater than that of the normal disk. The retina was torn on the nasal side of the opening and separated from the choroid for a short distance. There was a large hemorrhage in the upper part of the retina and numerous smaller hemorrhages scattered about the fundus. A few retinal vessels appeared to have a circulation of blood through them but the greater part were white and bloodless. Veins and arteries could be differentiated with difficulty.

In the course of a few weeks the excavation at the site of the nerve head was filled in with new-formed connective tissue, the hemorrhages were absorbed and the circulation restored in several of the vessels in the lower portion of the retina. The patient made a good recovery in all respects, except of course his vision, which was nil. His temperature ranged from 97° upon admission to 101°, reaching normal on the 28th day. He was later transferred to the State Asylum for the Blind.

Evulsion of the nerve is produced in two different ways, first and the more commonly reported, by the entrance into the orbit of a foreign body such as a sharp stick or the point of an umbrella, pieces of metal, stone, or shot. Here the foreign body impinges upon the nerve, making traction backward and at the same time forcing the globe forward. The pyramidal shape of the orbit and the facility with which the globe can be pushed aside contribute to the production of this form of injury. In the second group the separation of the nerve results through the application of a crushing force, driving the eyeball forward and over-extending the optic nerve until it is pulled out of the scleral opening. The case of Reis where the upper margin of the orbit was comminuted by the kick of a horse's hoof is a good example, and the subject of the writer's case perhaps especially well exemplifies this type.

Our knowledge of the changes in these cases is slight because the number of cases which have been studied from the

beginning and for some time after is small. The separation of the optic nerve manifests itself in the early stage by a complete loss of the papilla and its vessels. At the site of the papilla is found a deep excavation. Birch-Hirschfeld found in a partial separation after a penetrating wound an excavation of 6 D. (2mm) and Salzmann, after a shot injury a complete separation with a 4mm (12 D.) depression. The absence of the papilla shows as a gray or grayish blue hole. The retina may be more or less completely torn away, and if the latter, the retinal circulation is lacking. The choroid lies free, raises itself slightly, and the border of the separated retina shows a marked opacity. If the retina is not separated or only in a circumscribed place, then the retinal vessels are completely or entirely retained. The retinal circulation may be greatly increased by the proliferation of capillaries on the nervehead. Further, hemorrhages at the papillary border in the retina as well as at the macula appear. With partial separation, a deep excavation may remain behind or the excavation may be filled in by connective tissue growth, which piles up in a sheaflike deposit. Circumscribed pigment accumulation may appear.

As previously mentioned the separation of the nerve may be partial or complete. Birkhauser's patient suffered a penetration of the orbit by a cow's horn and with the ophthalmoscope he saw a small oval nervehead, with the long axis horizontal. The inferior margin was embraced by a very dark semilune which took the place of the missing portion of the nerve. There was a marked parallax between the center and the edge which left no doubt that the nerve had been partially torn loose in the lower quadrant of the scleral canal. The retinal vessels were lacerated in this direction and in six weeks the semilune had become replaced by new-formed tissue.

The dural sheath is rarely torn, as was demonstrated in the case of Dalen (*Ophthalmoscope*, vol. viii., p. 519). Microscopic sections of the eye enucleated because of glaucoma, two and one half years after the accident, showing a complete separation of the nerve but an intact dural sheath.

Experiments on cadavers were made by Hess in 1907 to produce evulsion of the optic nerve but with absolutely no success.

It would seem quite likely that in severe head injuries, such as occur in automobile accidents, train wrecks, etc., that a more careful examination of the eye ground or of the enucleated eye would show the evulsion of the optic nerve to be less rare than appears to be the case.

## OCULAR AFFECTIONS DEPENDENT UPON DISEASE OF THE TONSILS: TWO CASES.<sup>1</sup>

By DR. HOWARD F. HANSELL, PHILADELPHIA.

CASE I.—Mrs. W., aged 35, was admitted to the Jefferson Hospital, May 20, 1917, under the care of Dr. F. X. Dercum. She stated that in the night of May 16th she went to bed in her usual good health. On Thursday morning, May 17th, she awoke to find the right eye entirely blind and the sight of the left eye diminished. By Saturday she was blind also in this eye. She stated that on Thursday she could see one half of objects and this half was obliterated at the expiration of four days.

At the time of my first examination she had no light perception in any part of the field of either eye. The pupils were dilated ad maximum and did not contract under strong electric light or in association with convergence. Radiographs showed no abnormality of any kind in the bones of the cranium or the contents of the skull. The sinuses of the head and face were clear. The Wassermann test of the blood and the spinal fluid was negative; when the cord was tapped the fluid was forced out through the cannula slowly and showed no sign of increased pressure. It contained no blood cells or other cells indicative of any general or special etiology. The optic nerves were swollen and a few small superficial hemorrhages were seen in the retina adjacent to the disk of the left eye. The boundary of the nerve head and the lamina cribrosa were obscured by oedema. The oedema did not extend far into the retina. The left nerve was swollen slightly more than the right. At this time there were no hemorrhages in the right eye. Examination of the urine, quantity and quality, was negative. The heart and circulatory system and the abdominal organs appeared to be normal. The patient had no nausea or vomiting; no

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<sup>1</sup> Read at meeting of the American Ophthalmological Society, New London, July, 1918.

headache or dizziness; no pain. Indeed, the only complaint was of blindness.

She was of average height, rather fat and flabby. The only recent illness was tonsillitis, from a mild attack of which she recovered four weeks before admission into the hospital. For this affection she had been under the care of a physician.

Report of the examination of the throat by Drs. Chevalier Jackson and Fielding O. Lewis was that the tonsils were cryptic and contained broken-down tissue and probably pus, and ought to be removed. Up to this time the treatment had consisted of daily sweat baths, mercurial inunctions, and potassium iodide. No improvement whatever was apparent. Indeed, while the optic neuritis became no more pronounced, two or three hemorrhages had appeared in the retina adjacent to the nerve in the right eye. The pupils maintained their wide dilatation and light perception remained absent.

Twenty-four hours after both tonsils had been enucleated perception of light returned to the left eye, and in one day more to the right eye. The hemorrhages began to be absorbed and the swelling of the nerve head, never higher than one diopter, to subside.

During the eleven days of blindness no new symptoms developed. Mercury was badly borne and had to be discontinued. The iodide and the baths were kept up and strychnia was added. Each day after the tonsillotomy vision became better until it equaled counting fingers at six feet. The nerve heads have begun to grow pale on the temporal sides, the optic neuritis is passing on into atrophy. On June 13th daily applications of high frequency current were commenced. The return of vision has been gradual and not limited to any one part of the fields. Simultaneously the pupils began to decrease in size and the reaction to light was reestablished.

Without following in detail the recovery of vision it may suffice to state that it was gradual, and that in March, 1918, nine months afterward, it equaled R.  $\frac{6}{10}$ , L.  $\frac{6}{15}$ . At this time the optic disks were pale and their vascularity was reduced. The fields of vision were not abnormally restricted for white but slightly and concentrically for colors, all of which were slowly distinguished. During the first months of recovery she was treated with strychnia, potassium iodide, high frequency current, and massage.

After eliminating every other cause by careful and thorough investigation of the other organs of the body it was concluded that the source of infection was the purulent tonsils. The rapid loss of vision in each eye and the speedy recovery after enucleation of the tonsils seemed to point unquestionably to this etiology.

The onset and cause of the blindness do not correspond to the usual history of acute axial neuritis. In this affection pain is a constant symptom in the forehead and temple or deep in the orbit, increased by movements of the eyes or by deep pressure. Vision declines rapidly. The pupils are dilated and while they do not respond to light contract consensually. With increasing loss of vision the pain grows less. The blindness is seldom complete and binocular. The fundus remains normal or there is a moderate degree of optic neuritis. After several days of treatment by sweats or inunctions the periphery of the field is restored but a large central scotoma persists.

In perineuritis or neuritis interstitialis periphera, the central vision is preserved and the field is concentrically limited for white and colors.

CASE 2.—Mrs. C., aged 30, for several weeks had repeated attacks of iridocyclitis. In the intervals the eye cleared and pain subsided. Vision remained dull. For the last five days and nights the pain has been severe and uninterrupted and she has had but a few hours' sleep. Vision equaled  $\frac{3}{200}$ . The cornea was stippled, anterior chamber deep, and the iris partially attached to the lens capsule. The ciliary region was excessively sensitive to touch, and all the ciliary vessels were injected. Local and constitutional remedies had been ineffective. She was admitted to the Jefferson Hospital, and double tonsillotomy for purulent disease of the tonsils was performed by Dr. Fielding O. Lewis the same day. In a few hours the pain had entirely disappeared, the synechiæ were broken off, the pupil round, and the injection decidedly less. In twenty-four hours after the operation she left the hospital practically recovered. Ten days later scarcely a vestige of the inflammation remained. This was a most striking and sudden cessation of the pathologic process, indicating beyond question that the ciliary region was being constantly fed with toxic material from the purulent tonsils.

No bacterial examination in either case was made of the pus taken from the tonsils. The infection is probably not dissimilar to that caused by abscesses around the teeth for which the staphylococci are usually held responsible.

Steinbugler (ARCH. OF OPHTHAL., March, 1918), in discussing the mode of eye infection from a dental focus, which may be identical with that of tonsillar disease, refers to Leongt's view that it takes place through channels in the bone, sub-

periosteal tissue, or by way of the lymphatics; to Polet's view that in addition to these infection may be carried through the nerves; and Dutoit adds that of the ophthalmic vein and cavernous sinuses. He asserts that a direct extension through the venous channels from the diseased area to the cavernous sinuses is anatomically possible and unquestionably has produced a sinus thrombosis and its symptoms.

E. V. L. Brown (*Ophth. Rec.*, June, 1917) reports a case of sympathetic iridocyclitis and choroiditis with preservation of useful vision in the sympathizing eye. The enucleated left eye showed typical changes. The right had thickened, tumefied iris with precipitates. Marked improvement did not occur until infected tonsils were removed, when vision improved from counting fingers to  $\frac{4}{10}$ , and has retained this acuteness for fourteen months.

Other cases, notably those reported by Brown and Irons (*Am. Oph. Soc.*, 1916), to the number of sixteen out of one hundred, point to the same etiology. Zentmayer (*Am. Jour. Ophthal.*, Apr., 1918) says: "That the tonsils have not more often been found to be the source of the ocular infection is surprising, when we consider how frequently they are blamed for general infection." The answer to this comment lies in the ignorance and carelessness of the oculist. In view of the great importance of focal infection in ocular disease to which the attention of the profession was called by de Schweinitz in his masterly paper read before the Ophthalmological Section of the International Medical Congress held in London in 1913, it becomes our plain duty to investigate the condition of the tonsils in every case of intraocular inflammation.

William Lang, "Etiology and Treatment of Iritis" (*Lancet*, June 23, 1917). Ten cases in private practice. In six cases there was disease of the tonsils.

I would refer those particularly interested in this subject to the paper of Dunn of Richmond, Va. (*ARCH. OF OPHTHAL.*, Sept., 1917), who describes a number of cases of uveitis due to tonsillar disease and relieved by treatment directed to the tonsils. Such papers impress us with the necessity of diligent attention to the tonsils in inflammations of the uveal coat that are not venereal, tubercular, or traumatic.

## SOME OBSERVATIONS ON THE RESTORATION OF THE ORBITAL SOCKET.<sup>1</sup>

BY DR. P. N. K. SCHWENK AND Dr. WM. CAMPBELL POSEY,  
PHILADELPHIA, PA.

*(With seven illustrations on Text-Plates XIX. and XX. and five cuts in the text.)*

THE restoration of a socket of sufficient depth and of such configuration that an artificial eye of suitable shape may be properly fitted into it, is never an easy task, particularly when the occlusion of the socket is complete and no conjunctival lining of the lids remains. It is not the purpose of this paper to enumerate all of the methods which have been devised by operators for the accomplishment of this purpose, or to enter into comparison with the excellent results obtained by Weeks and others by the employment of Wolff and Thiersch grafts. These operations are well known and properly appreciated. The case, however, with which the lower cul-de-sac may be restored by the ingenious transposition of the skin of the cheek subjacent to the lid, into the floor of the orbit, as practiced by Maxwell, led the writers to a combination of this method with a utilization of a skin flap taken from some point above the orbit for the formation of the upper cul-de-sac. Zentmayer has successfully utilized Maxwell's procedure in forming the upper cul-de-sac, but this operation is complicated by the presence of the levator, and is not as easy of performance, nor are the results as satisfactory as when applied to the lower lid. All who have practiced plastic surgery about the orbit have experienced the rapidity with which healing occurs in this region, and the adaptability and vitality of flaps trans-

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<sup>1</sup> Read at meeting of the American Ophthalmological Society, New London, July, 1918.



planted from parts adjoining. An exact planning of the incisions, and a precise coaptation of the margins of the flaps to the tissues with which they are brought in contact, insure prompt healing, and after a time an often undetectable scarring.

Our method of procedure is as follows: A careful separation of the margins of the two lids from one another and a free dissection of the tissues sublying. Then follows a division of the external canthus. The lower cul-de-sac is then restored by Maxwell's method and the upper by transposing a long flap taken from the skin of the forehead above the brow, care being taken, however, that no hairs of the brow are included in the margin of the flap. The edges of the flap must be nicely coapted to the margin of the upper lid, and the upper border of the skin secured by the Maxwell procedure. The next step is of great importance, namely, the restoration of the external canthus, for unless this be accomplished at this time, the conformer which is introduced between the lids at the completion of the operation will not be retained in the cul-de-sac, and a subsequent operation for the restoration of the canthus will be necessary. To secure a firm canthus, two double needled sutures of No. 4 silk are passed through the skin of the upper pillar of the old canthus and brought out through the raw surface of the pillar; they are again reunited into the raw surface of the lower pillar, and made to issue upon the skin surface of that structure. They are then finally tied on two pearl buttons. The pedicle of the flap is thus buried under this bridge of tissue, and unless the tension upon the pillars is too great, which may be avoided by a careful dissection of the tissues of the canthal pillars prior to inserting the sutures, union is prompt and satisfactory. Should the tissues give way, the canthus may be restored later, after the flaps have united, by excising a diamond-shaped area of skin from the pedicle of the flap and uniting the edges of the denuded area in the manner just described.

Some of our colleagues have criticized our method of canthus repair, saying that a sinus would result from the burying of the skin flap under the bridge formed by the union of the two pillars. As a matter of fact, however, this has not occurred, and in the three cases about to be reported union was firm and there was no trace of a fistulous tract. Nor were we both-

ered by outgrowth of hairs from the flap. This may probably be accounted for by the youth of the patients and by the absence of hair from the tissues whence the flaps were derived. During the first few months there was considerable desquamation of skin from the flaps, but this gradually subsided and the sockets became dry and more and more commodious. A point to be accentuated is the importance of retaining the conformer in the socket for a long time, interfering with it as little as possible. The conformer being of the same form as an artificial eye, gradually molds the orbit into the shape necessary for the retention of the prothesis. A firm bandage should be kept constantly applied over the orbit for at least two weeks. The stitches inside the cul-de-sac should be removed at the end of a week, those from the canthus in eight or ten days.

CASE I.—J. A., aged 20 years, applied to the Wills Hospital for the restoration of the left socket, which had been obliterated as the result of a lime burn, as a consequence of which the burned globe had been removed some years previously. Examination showed complete anchyloblepharon, and it was impossible to insert even the smallest probe between the firmly united lids. The skin of the lids and the surrounding tissues was uninjured. The patient was etherized and after the lids had been carefully dissected apart, the external canthus divided, and the sublying tissues freely undermined, the lower cul-de-sac was restored after the method of Maxwell. A long pedicled flap was then obtained from the forehead above the brow, carefully sewed into position in the upper part of the wound, and a conformer inserted into the socket thus obtained. The canthus was then restored in the manner already described and a bandage applied.

Healing was prompt. As the patient lived at a distance, he was permitted to go home, with the conformer in excellent position. Unfortunately, however, the oculist to whom he was referred, not appreciating the importance of careful oversight over the patient, permitted him to become careless in the flushing out of the socket with boracic acid solution, in consequence of which the nasal end of the conformer slipped out of the socket and a rapid contraction of the newly found cavity resulted. Upon his return to us, it was found that the nasal sulcus of both the upper and lower cul-de-sacs had contracted, and the conformer was no longer retained under the pillars at the outer canthus. Subcutaneous incisions were made under local anæsthesia along the

lid borders and under the tissues at the canthus, and double armed sutures buried in the apices of the cul-de-sacs, being brought out upon the skin surface and tied over buttons, about 4 or 5mm from the ciliary borders of the lids. The conformer was again reinserted and the patient kept under close observation for a month. At the expiration of this period, a small segment of skin was excised from the somewhat redundant base of the flap, where the pedicle had been twisted in turning the flap into the orbit. An artificial eye can now be worn with comfort and the cosmetic result, apart from the immobility of the eye, is perfect.

CASE 2. R. B., male, aged 21 years, right eye and conjunctival cul-de-sac badly burned by lime in November, 1917. After enucleation, dense cicatricial bands almost entirely obliterated the socket, the inner portion of the lower cul-de-sac alone having any appreciable depth.

Ether was administered, and the lower cul-de-sac restored by Maxwell's method. Some weeks later, the external canthus was divided under ether, the upper lid freed from its cicatricial connections by dissection, and a flap which was taken from the skin of the upper lid just below the brow superimposed upon the raw surface thus obtained. The canthus was restored by the method just cited after a conformer had been inserted into the amply spacious socket. On the fifth day, however, the stitches holding the pillars of the canthus in apposition gave way, necessitating a refreshing of the edges of the flap and a reunion of the pillars some time later. Union was now permanent and a suitable sized eye is worn with comfort.

CASE 3. Male, aged 18 years, lost his right eye from a lacerating wound, demanding enucleation. In consequence of faulty technique in this operation, the upper lid was firmly bound down to the sublying tissues by dense cicatricial bands. The lower cul-de-sac was also somewhat contracted.

Ether was administered, the external canthus divided, and the lid freed of its cicatricial connections by dissection, the conjunctiva being undermined uniformly to the margin of the lower lid. The lower cul-de-sac was then formed by the method which will presently be reported, leaving a raw surface in the upper half of the socket. This was covered by a large pedicled flap taken from the skin of the upper lid just below the brow, as in the preceding case. After careful union of the edges of the flap to the surrounding tissues, and deepening of the apex of the cul-de-sac by three double mattress sutures, which were passed through the skin flap about 4mm from the lid margin and brought out through the skin of the brow about 6mm from the ciliary border, a

conformer was inserted and the canthus re-established as in the two previous cases. The raw surface marking the site of the flap was carefully covered by uniting the edges of the wound and a firm dressing applied. Recovery was uneventful, and a proper sized eye is retained without difficulty.

In the opinion of the authors the Maxwell operation is admirably adapted for the restoration of the lower cul-de-sac, as repeated trial has proved. The one objection to the procedure, however, is the tendency that the lid evinces to ectropionize at its outer half after healing has occurred. This may be partially overcome by making the flap narrower by 2 or 3mm than recommended by Maxwell, especially at its outer portion. The authors have not experienced the puckering in the skin below the lid, mentioned by some writers, except in one instance, where the deformity was readily overcome by excising a small wedge-shaped portion of skin, with the base up, at the point of greatest skin redundancy. As the operation seems to be but little practiced in this country, the authors have thought it might be not without interest to describe the various steps.

#### MAXWELL'S OPERATION.

Instruments—Scalpel, dissecting forceps, pressure forceps, glass mask, needles, and needle-holder.

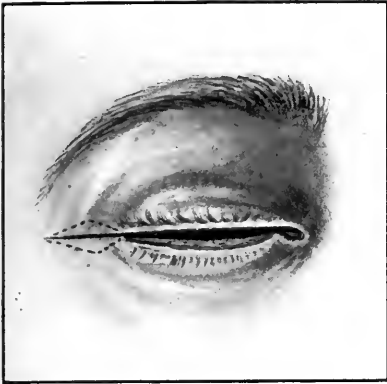
*First Stage.* An incision is made at the bottom of the shallow cul-de-sac in its whole length, some 5 or 6mm deep. It is made the same depth along all its extent (a, b, in the diagram).

*Second Stage.* On the skin of the lid are made two incisions, one about one sixth of an inch below the lid-margin and the other curved below this, enclosing between them a semilunar area of skin about 12mm broad in the widest part. The length of this curved incision (c, d, in the diagram) must be greater than that of the first. The crescent of skin thus marked out is used to form the lining of the new cul-de-sac.

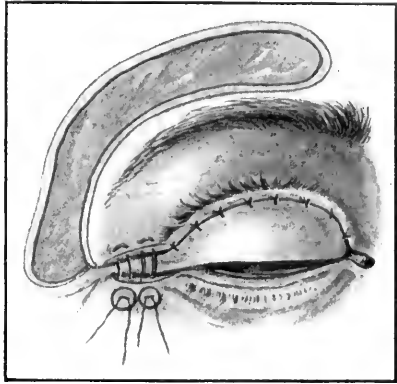
*Third Stage.* The upper limiting incision of the crescent is deepened until it joins the incision in the cul-de-sac. The margin of the lower lid now forms a bridge attached only by its two extremities.

*Fourth Stage.* The crescent of skin is dissected up from its

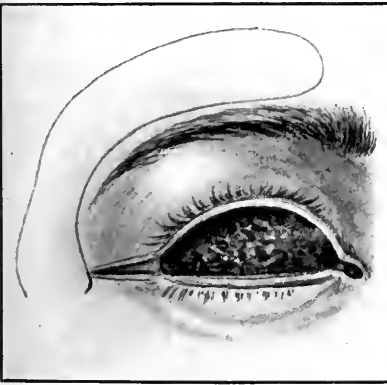
ILLUSTRATING DRs. SCHWENK'S AND POSEY'S ARTICLE "SOME OBSERVATIONS ON THE RESTORATION OF THE ORBITAL SOCKET."



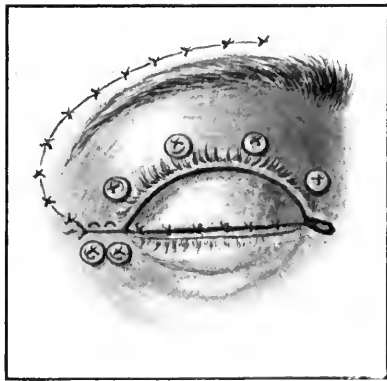
A



C



B



D

FIG. 1.—(A) Division of external canthus and separation of the lid margins; (B) outline of pedicled flap; (C) transposition of flap from forehead into upper portion of cul-de-sac, and restoration of external canthus; (D) coaptation of flap margins to parts adjoining.



ILLUSTRATING DRS. SCHWENK'S AND POSEY'S ARTICLE "SOME OBSERVATIONS ON THE  
RESTORATION OF THE ORBITAL SOCKET."



FIG. 2.—Case 1. Restoration of canthus.



FIG. 3.—Case 4. Before operation.



FIG. 4. Case 4. After operation.





borders until only a relatively small part (about one sixth of the whole) near the center remains attached to the subjacent tissues. This is represented as unshaded in the diagram.

*Fifth Stage.* The bridge of skin bearing the lashes is held forwards, and the crescent slipped under. The two extremities are secured into the angles of the first incision, and to its posterior lip the upper border of the crescent is fastened by two or three sutures. The lower border of the crescent is then brought up and stitched to the conjunctival edge of the bridge. The crescent is thus doubled on itself, and forms a groove, the posterior wall of which is made by the upper,

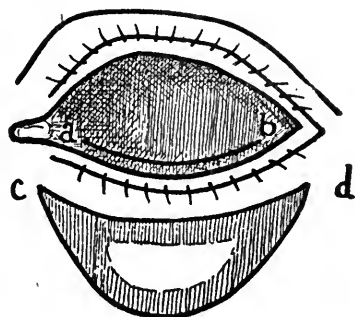


FIG. 5. Incisions for the Maxwell operation.

the anterior by the lower half of the crescent. The undissected pedicle holds the floor of the groove down.

*Sixth Stage.* It remains to close the wound on the surface of the lid, and this must be done carefully or there will be a troublesome scar. The two edges are very disproportionate in size: the lower much longer than the other. The first step towards closing the wound must be a stitch in the center of both edges, then one on each side midway between the center of the extremities. The coaptation of these two edges is the most difficult part of the operation.

This completes the whole procedure, but, before the pad and bandage are applied, a glass mask, preferably with a central opening, must be placed within the lids to depress the new-formed cul-de-sac until healing is advanced. It should be left in for a week or ten days: the central opening is an advantage, as it enables the socket to be washed out easily.

Another ingenious method for restoration of the socket has been proposed by Golovine. In cases of exenteration of the orbit, this writer endeavored to supply a socket suitable for the reception of an artificial eye, by slipping a tongue of skin taken from the temple under a bridge of undisturbed skin at the outer aspect of the orbit. His plan of procedure was as follows: A cutaneous flap A is first shaped from the temple, as shown in the figure. A semicircular incision, a, b, is made and continued to c, through a subdermal opening, that com-

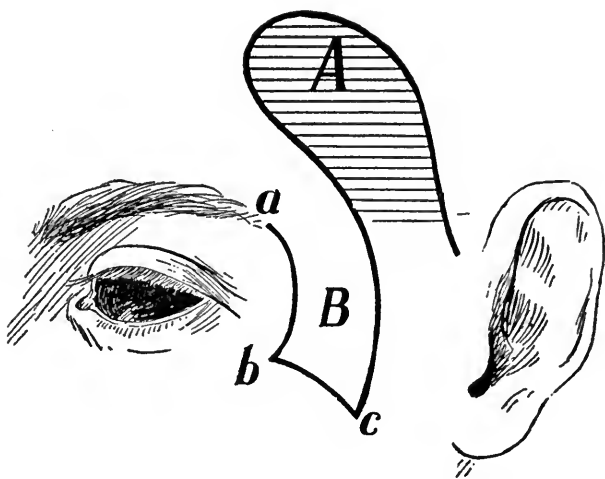


FIG. 6. Incisions for the Golovine operation.

municates with the orbit along a, b, the upper rounded edge of the large flap is slipped under, and the remaining tissues rearranged as in Fig. 7. The sliding flap applied and arranged within the orbit (epithelial surface outwards) is carefully stitched to the cut edges of the remaining conjunctiva and so a space is formed in which the prothesis may rest.

In cases where the occlusion of the socket is incomplete, some of the conjunctiva being still present, flap transplantation is not always necessary and other methods are preferable. Wiener's method is especially applicable to this class of cases, as the authors can affirm after its successful trial in several instances. As some years have elapsed since its publication,

the various steps are given in Dr. Wiener's own words, as follows:

"My method has its use where the socket, while shrunk, has still a small amount of conjunctiva left. An incision (A, B, C, Fig. 8) is made through the conjunctiva, and a flap, including only the conjunctiva, is carefully dissected down to the lid margin (A, C, Fig. 8). This dissection after being started with a knife can easily and quickly be finished with a small curved blunt scissors. The dissection is then continued with the

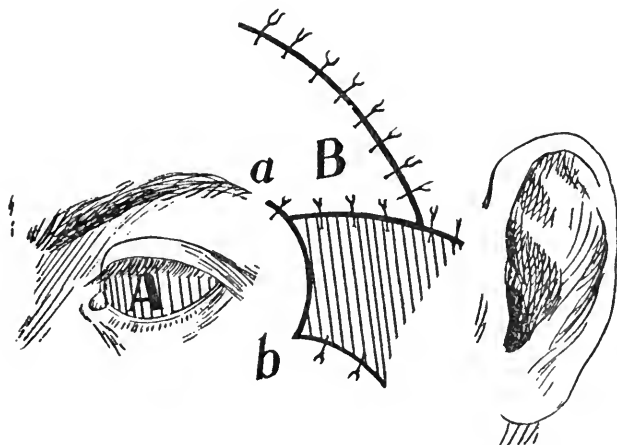
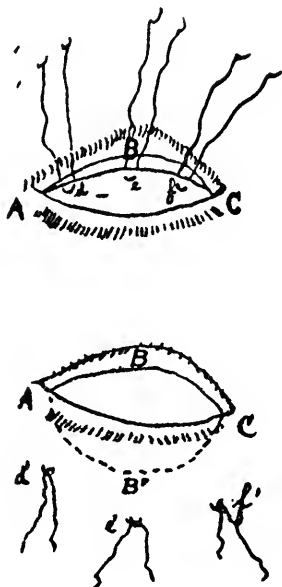


FIG. 7. Closure of the skin incisions, Golovine operation.

scissors so as to loosen the skin below the lid margin (A, C to B, Fig. 9), leaving a raw surface toward the bulbar side extended from B to B' and A to C (Fig. 9), and on the palpebral side A, B, C (Fig. 9). Then sutures with a needle on either end are introduced at the points d, e, f (Fig. 8), and passing them through the bottom of the newly made sulcus are brought through the skin at d', e', f' (Fig. 9) and tied over a button. This gives a conjunctival covering for the lower lid and leaves the bulbar surface A, B, C, B' (Fig. 9) to be covered. This is done by covering a lead plate, previously shaped, with grafts from the thigh and placing carefully in position. A dressing is then applied and both eyes bandaged, the patient remaining undisturbed in bed for four days, when the outside dressings are replaced by clean ones without disturbing the plate. On

about the eighth day the plate may be removed, cleaned, and replaced. The glass eye can usually be worn by the tenth or fourteenth day. The stitches at d', e', f' (Fig. 9) are not removed, but are tightened each day after the fourth day until they pull through. This is an additional help to holding the lower sulcus intact, as the internal scars resulting tend constantly to pull on the bottom of the sulcus and thus heighten



FIGS. 8 and 9. Wiener operation.

the effect. It is important that these stitches should be allowed to pull through and not be removed before, for one is tempted to remove them after a week when the lower sulcus may appear too deep. I have made this mistake, thinking I had secured a lower sulcus of exaggerated depth, when, on removing the stitches, it rose too high and became almost obliterated, so that the stitches had to be replaced."

In 1915, one of the authors of this paper (Dr. Schwenk) without knowledge of Dr. Wiener's contribution, in an attempt to restore a shrunken socket, where some mucous membrane was still present, proceeded as follows:

The conjunctiva was undermined and brought forward, doubled upon itself to form a new cul-de-sac. To do this the conjunctiva was entirely loosened from the floor of the orbit from a point just below the margin of the lower lid to a point 3 or 4mm above the nerve head, by means of scissors, curved on the flat, entrance being made at the outer canthus. A transverse band of skin about 6mm wide, having its upper limits 3mm from the lid margin, was undermined from canthus to canthus by means of a Graefe knife inserted at the temporal side. By means of the scissors the remaining tissues of the lid were then cut through transversely into this subcutaneous pocket at a point 3mm from the lid margin, thus increasing the depth of the orbital cavity by an amount equal to the depth of the subcutaneous dissection. Three double-armed heavy silk mattress sutures were inserted in the loosened conjunctiva from above at a point 7mm from its attachment at the lid margin and the needles were carried into the subcutaneous cavity, where they were passed out through the skin at its lower part and tied on Ziegler pearl buttons; a gold conformer was then inserted.

Considerable hemorrhage resulted from cutting the hypertrophied conjunctiva, but there was no subcutaneous ecchymosis, and healing resulted without undue reaction. The stitches were allowed to remain seven days when the conformer was replaced by a glass eye.

The operation is indicated in cases in which the conjunctiva is sufficiently redundant to allow of its being slipped into the anterior cavity. It differs from the Wiener procedure in the location of the cul-de-sac anterior to the cartilage of the lid, instead of posterior to it. In the Wiener operation a large denuded area from which the conjunctiva has been taken is left exposed in the socket, which must be covered with Wolfe or Thiersch grafts. In the author's method, no raw surfaces remain, as all the dissection is done beneath skin and conjunctiva. The following case is reported as illustrative of this method.

CASE 4. M. L., male, aged 28 years. Right eye had been enucleated 11 years before on account of severe injury in a bottling establishment. Badly fitting cheap glass eyes

were worn for many years, until cicatricial contraction of socket prevented their retention. Examination showed the conjunctiva to be much thickened, corrugated, and filled with cicatrices.

Operation under ether as follows: By means of a slightly curved-on-the-flat scissors, the conjunctiva was entered at the outer canthus and two thirds of orbital mucous membrane was undermined, cutting cicatrices for some distance above the apex of the orbit and anteriorly to the margin of lower lid. Hemorrhage was pronounced, but was controlled by applications of adrenalin. A Graefe knife was then inserted just below outer canthus, and a broad band of skin was loosened and undermined, about 3mm from margin of the lid and 3mm wide parallel to its edge, across the entire width of lower lid. The submucous and the subcutaneous cavities were then united by subcutaneous incision across whole lid. Three double-armed silk mattress sutures were then inserted into the loosened conjunctiva 6mm from lid margin, the conjunctiva lifted from its bed on the floor of the orbit and carried with needles to the apex of subcutaneous cavity and passed through the skin externally and each tied over a pearl button to avoid puckering of the skin. The conjunctiva was carefully adapted to the lid and floor of orbit and stitched to orbital tissue to avoid sliding or displacement. The stitches thus inserted served as anchoring points and encouraged union. A gold conformer was inserted and a firm dressing was applied. The stitches were removed on the 8th day. Recovery was uneventful and the result all that could be desired, a proper sized prosthesis being worn with comfort. Fig. 4 shows condition 2½ years after operation.

UNILATERAL INHERITANCE. LOSS OF THE HEREDITARY CORRELATION BETWEEN THE TWO HALVES OF THE BODY, THE PAIR ORGANS, AND ESPECIALLY THE EYES. DESCRIPTION OF TWO CASES OF THE SO-CALLED PHYSIOLOGICAL ANISOCORIA.<sup>1</sup>

By DR. ANTON LUTZ, HABANA.

(With six cuts in the text.)

BEAUTY is the expression of health, and is almost always based on a complete harmony of the different parts of the body. This harmony gives the impression of tranquil majesty which we receive upon contemplating ancient sculptures, such as Ludovisi's *Hera*, Otricoli's *Zeus*, Ludovisi's *Ares*, or the bust of Asclepius. We get the same impression when admiring the works of the renaissance idealistic school, of which I recall the *Mona Lisa* of Leonardo da Vinci, the *Madonna of Brügge*, of Michael Angelo, and especially the *Sistine Madonna* of Raphael. On the other hand, the Netherlands realistic school very often takes advantage of an asymmetry to obtain a humorous effect, as, for instance, Teniers in his *Dentist* or his *Alchemist*. Michael Angelo uses very seldom a complete harmony, preferring asymmetries to represent vigorous movement in his works. In the same manner, caricaturists exaggerate the asymmetry of the human body to produce a ridiculous effect. This may also be found in portraits of abnormal personalities; notable examples are: the self-portrait of the Spanish author Greco in his painting *Count Orgas's Funeral*, and the bust of the Grecian philosopher

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<sup>1</sup> Presented at the Fourth National Medical Congress in Cuba, 1917.

Esopus, but more especially in the portrait of Saint Peter the Apostle, in the Saint Marcellin catacombs.

Besides there exists a certain antipathy for persons with marked asymmetry, for instance, of the face; they are looked upon as inferior beings, and the physician declares this asymmetry to be a sign of degeneration. A complete symmetry or harmony is very rarely found in nature. It is the origin of beauty, and it is a significant fact that divinities of all ages (even in Egypt and Babylon) have always been represented very symmetrically. This brings to our memory Raphael's words when he said that models were quite useless to him, since his works corresponded almost solely to his mental conception of beauty. Among men the rule prevails of a slight asymmetry, which may be proven by pictures of persons of all times, for example, the statues of Solon, Democrates, Sophocles, Demosthenes, of Goya's mother, etc.

Anthropologists have shown that race plays a great part, so much so that the European race shows, for example, the asymmetry of the cranium much oftener and in a more marked degree than the black race (Martin, *Anthropologie*, 1913). The reason of negroes and the insular peoples of the Pacific showing a greater symmetry of the body may be explained perhaps by the fact that they have lived during thousands of years isolated and have not had the occasion of crossing with nearby foreign elements.

Criminologists have made a special study of asymmetry of the face and of the cranium, which is to be found so often in criminals (Lombroso deems the face asymmetry as an important mark of degeneration and gives in the atlas of his work, *L'uomo delinquente*, 1888, some truly amazing instances of asymmetry of the head, also mentioning the busts of Nero and Messaline), and they have been able to verify that different sides of the face correspond sometimes exactly to those of the two parents or to those of two different individuals in the ascendancy. They took a photograph of the face as seen from the front; they divided it half by half, and completed each half with its corresponding image reflected in a mirror, thus obtaining two distinct physiognomies, which resembled those of individuals in the ascendancy. It seems very natural that such observations be found especially in the criminologist



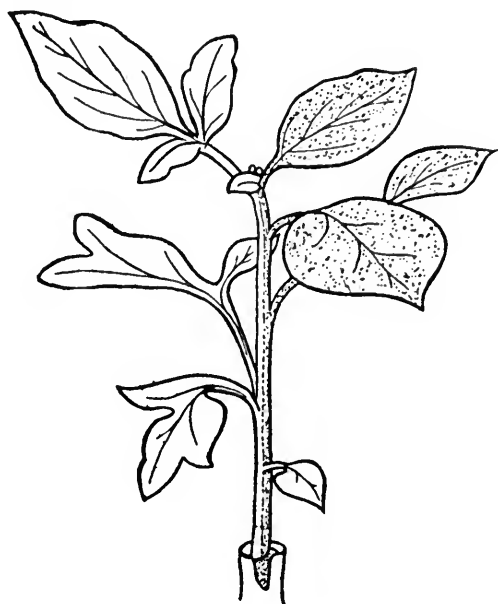
literature which has time and material at its disposal. But such observations also exist in medicine. Bleuler (*Naturforschende Gesellschaft Zürich*, 1911) refers to a sage friend of his whose face corresponded on one side to that of his father, and on the other side to that of his mother.

In considering these observations the following question arises: Does a unilateral inheritance exist in such a form that one half of the body may differ in whole or in part from the other half, and due only to hereditary agencies, that is to say, transmitted by the same germinative plasm, and not occasioned by mechanical or toxic agents during the development? To solve this problem we have two ways open (not considering experiments in the vegetable and animal worlds) namely: (1st) the photographic method, for instance, of the two halves of the face; (2d) the genealogical investigation, that is, by searching for unilateral presence of an anomaly doubtless inherited during several generations past, in the same parentage.

1st. In so far as I know, there does not exist any reliable observation on the asymmetry of the entire body occasioned solely by inherited agencies,—for instance that one half of the body corresponds to that of the father and the other half to that of the mother. But we can expect to find some day such a chimera. This hope is based on the splendid experiments of the botanist, Winkler (1907) in Tübingen, which are of the utmost importance for biology, and deserve to be known to every physician. He crossed by means of grafts two different varieties of a plant (*solanum nigrum* and *solanum lycopersicum*) and thus obtained a specimen which corresponded in its branches partly to the *nigrum* and partly to the *lycopersicum*, and having leaves one half of which were *nigrum* and the other half *lycopersicum*.

The zoölogist Arnold Lang (Zürich, 1910), did not believe it impossible to obtain experimentally (influencing the blastomeres period) animals who should correspond on one side to one variety of a frog, and on the other side to another variety of the same species. Furthermore we have a series of observations on congenital asymmetry of the entire body. Cusson gathers in his thesis (Paris, 1905), twenty-six cases of hemihypertrophy of the body; among these some are found to be

without the slightest sign of disease (elephantiasis, neurofibromatosis, vasculosis, vasometric anomaly, etc.). A microscopic



*Solanum*  
*lycopersicum*



*Solanum*  
*nigrum*

FIG. 1.—Sketches by Winkler. Taken from Arnold Lang, *Über Vererbungsversuche*, 1909.

examination showed a larger size of the ectodermal and mesodermal elements, but without the slightest pathological change, the hypertrophic half corresponding exactly to that of a normal man of larger proportions. In the description the

examination on unilateral inheritance is lacking. Of interest also are the observations of Ives, who gathered in his thesis, *Contribution à l'étude de l'œdème unilateral* (Paris, 1902), a series of œdema cases of reflex origin, which were limited to only one half of the body. The hereditary character of these œdemata has been proven many times. Two forms are known: the *acute*, of Quinke, of which Osler saw the hereditary transmission through six generations, from one to the next; and the *chronic*, or *congenital*, of Meige, of which Milroy has given us a genealogical tree of six generations (*vide* Ouvry, Thèse, Paris, 1905: *Les œdèmes familiaux*).

Magnier has in addition called attention to the fact that some hypersensitive people show on one side of the body a much greater vulnerability than on the other. He collects in his thesis (*Réactions morbides unilatérales, localisées à un seul côté du corps*, Paris, 1915) a series of observations on persons in whom the organs of one side of the body showed a tendency to sickness doubtless much greater than those of the other side.

In view of these experiments and calculations, we may hope to find some day the mulatto, who on one side of his body shall appear European and on the other African. Thus he should represent the unilateral inheritance of the characteristics of the race. One physician, a globe-trotter, who had himself a museum of medical curiosities told me that he was quite sure of having seen about thirty years ago in a medical museum at Bordeaux, a child born in due time, who showed one half of his body with the signs of the negro race and whose other half was completely white. I was not able to get more exact information about it. One observation which very nearly approaches furthermore such a probability is the following: A white girl gave birth to female twins, one of which showed all the features of the Caucasian race, whilst the other had all those of the black race. It was proven by investigations that the father of the twins was probably a negro, which conforms with the theory that both twins were originated by the same ovum, fecundated by two spermatozoa at the same time.

2d. Upon examining the different organs of the human body for unilateral hereditary anomalies, we have to consider first the skin:

A. (a) Albinism is 'an anomaly which is inherited in the animal and vegetable worlds in the recessive way. This leads us to hope that we may some day find Europeans, Africans, and Asiatics, who will show only one half of their body albinotic. I do not know whether there exists any reliable observation on this point. In man, observations on albinism are, in a certain degree, more difficult than in animals, because albinism is not always complete, but approaches somewhat to the light type. Furthermore, recent investigations have shown that in albinism the pigment is not only lacking in the skin, but also in the nervous system, the sympathicus, and the brain (substantia nigra). These discoveries are important for the explanation of the nystagmus, which almost always accompanies albinism of the eyes, and moreover tends to demonstrate that albinism is not only an anomaly of the skin, but of the whole ectodermal layer.

(b) Ichthyosis of the skin is observed as albinism in various persons of the same family, to wit: that it is also inherited in the recessive form (there being also cases of dominant inheritance); and we have observations on unilateral ichthyosis.

(c) Hypertrichosis is to be inherited in the dominant form, to wit, directly from the grandfather to the father, to the son, to the grandchild, etc., and normal members of the family have normal descendants. An excellent example is the Siamese family. We are still lacking observations on unilateral presence.

(d) The odor of the skin is not only characteristic of the human race, but even of all parentage. Ch. Féré calls attention (*Revue de médecine*, 1902), "l'Hérédité de l'odeur," to the point that, for instance, dogs are never mistaken in recognizing members of the same family. He cites further Hammond's observation ("The Odor of the Human Body as Developed by Certain Affections of the Nervous System," *The Medical Record*, 1877), of an hysterical woman who during her attacks perspired in only one half of her body, which unilateral perspiration emitted a violet smell.

B. Another apparatus showing hereditary anomalies is the skeleton.

(a) A hereditary anomaly whose transmission has been observed during seven generations is the polydactylia. Struthers (Edinburgh, *New Phil. Journal*, July, 1863) refers

to the following tree: "In the first generation a supernumerary finger appeared on one hand; in the second generation, in both hands; in the third, three brothers had supernumerary fingers on both hands, and one of them had also a supernumerary toe on one foot; in the fourth generation polydactylia existed in the four extremities. Unilateral polydactylia has also been observed in the cat, chicken, and horse.

(b) Drinkwater and Farabee have described a new hereditary anomaly of the skeleton which they call brachydactylia, and which consists in the absence of the first phalanx in the fingers, so that all fingers have the same structure as the thumb. Not having the original accounts at my disposal, I cannot tell whether this anomaly, which has been seen transmitted through seven (respectively five) generations, appears also on one side only.

(c) On unilateral inheritance of anomalies of the skeleton of animals. I have found some notations in Darwin (*Variation of Animals and Plants under Domestication*). He recalls Hofacker's observation, who in 1787 saw in a German forest a deer with only one horn; in 1788 two, and later on several deer were observed having but one horn on the right side. Darwin mentions also that very often sheep are to be found having on one side one horn and on the other side two horns. In the same work we find Anderson's observation on a bitch dog who had only three legs, and which bred several pups, all with the same deficiency. Anderson also recalls a genealogical tree of rabbits having but one ear.

(d) Odontology possesses as yet very few observations on hereditary transmission of anomalies. Sometimes aplasia of a tooth is inherited. Magitot (*Traité des anomalies dentaires*) saw a lady lacking from birth both lateral superior incisors, and the same defect was to be found in her daughter and in her grandchild. Quellen, M. (*Dental Cosmos*) observed the hereditary transmission of the same imperfection through three generations (grandfather, mother, and daughter). Galippe, V. (*Revue de médecine*, 1901, "Études des anomalies dentaires") observed the same imperfection, but only on one side, and also in three generations. Furthermore, he saw in a lady congenital bilateral lack of both superior incisors, whilst her daughter lacked but one.

(e) Musculature. The hereditary sickness best known of the musculature is the pseudo-hypertrophia muscularis whose hereditary character has been recognized by its first describers. Eichhorst has given us a magnificent genealogical tree of six generations (*Berliner klin. Wochenschr.*, 1873). Hoffman saw it in twin brothers, children of the same mother and same father. Nicalaysen (Virchow's *Jahresbericht*, 1876) saw it in two half-brothers, children of the same mother.

Gowers (*Lancet*, 1879) saw twin brothers of whom only one had inherited the sickness. I have been unable to find a unilateral case of this sickness. A disease still more rare of the musculature represents Thomsen's congenital myotony. Also of this we may expect to find some day a unilateral form.

C. Senses.

1. Smell. Abundo has described a hereditary form of anosmia, which is almost always mixed with hereditary hypogustation (*Rev. neurologique*, 1894); Eichhorst says that this is to be found also unilaterally (aplasia nervi olfactorii).

2. Eyes. No one has so many facilities to study the loss of correlation between the two halves of the body as the eye specialist, and consequently it is in the ophthalmological literature that we find the larger number of observations on unilateral inheritance.

(a) The most striking is perhaps the loss of the correlation of the iris color called heterochromia which had already attracted the attention of C. Darwin. In two earlier works (Lutz, Anton, *Zeitschrift f. Augenheilkunde*, 1908, and *Deutsche medizin. Wochenschr.*, 1910) I could verify Gunn's and Sym's presumption, based on a series of twenty-five cases, that the ascendance of individuals with heterochromia iridum always shows a crossing between two different colors of the iris. I could furthermore demonstrate in four families, that the child with heterochromia formed the transition between sisters with dark-colored eyes of one of the fathers, and those with the light eyes of the other of the parents. This circumstance that the child with the asymmetry is found as a transition among symmetrical children contrasts with an ancient observation based on statistics (Orchansky) to the effect that the fourth or fifth child always results better than the previous or the following ones, the loss of the correlation being doubtless an anomaly

or even a sign of degeneration. Sym (*Ophthalmic Rev.*, 1889) observed heterochromia in a child whose father had light eyes, but in one parentage all had dark eyes. In a twin child (of two ova) I observed that the father had blue eyes and the mother brown eyes, whilst one child had on one side a light brown iris and on the left a dark brown iris; the twin sister had blue irides in both eyes. Fuchs observed in twin sisters (of the same ovum) marked heterochromia: one of the twin sisters had the dark color of the father's eyes on the right side, and the light color of the mother's eyes on the left; the other twin sister showed the heterochromia iridum inversely (Fuchs, *Zeitschrift f. Augenheilkunde*, 1906). Przibram (*Archiv f. Entwicklungsmechanik*) could transmit experimentally in cats heterochromia iridum through various generations; the correlation between the light eye and deafness on the same side, which is to be found in cats, was not interrupted.

(b) Another loss of the correlation between the two eyes can be verified many times in anomalies of the refraction in anisometropia. Every eye specialist has seen cases in which one eye was emmetropic and the other one was markedly myopic or astigmatic. In four cases I was able to verify that the hypermetropia in one of the eyes corresponded to the father's eyes, and the myopia of the other eye to the mother's eyes, or inversely. The concordance of the anisometropic eyes was so very exact that they corresponded to one half of a diopter to the corresponding eyes of the parents, and one could really say that the patient had inherited in one eye the visual refraction of the father, and in the other one that of the mother. So far as I know, we have not yet a large series of observations on the relation between the anisometropic eyes of a person and those of his parents (Steiger's great work is not at my disposal), but every eye specialist will have occasion to add such new observations. These cases of marked anisometropia (for instance, hyperopia + 3.00 diopters on one eye and myopia -4.00 diopters on the other, as was shown in one of my patients) also show clearly that in the development of refraction in man, we have chiefly to blame hereditary influences, and that all other factors to which the cause was attributed have only the importance of favoring circumstances, such as working at close range of vision, an inclined position,

congestion of the cranial vessels by reason of tight collars, etc. They may be compared to the influence of climate on general pigmentation: thus, the tropical climate gives clear evidence of southern ancestors in the northern Europeans who live a long time in the tropics. If we wish to explain the development of myopia by a long pupillary distance, by the orbit's formation or by the thinness of the sclerotic, we need only state again that the condition of refraction is caused by hereditary features of the race, since specifically as to orbits we know through

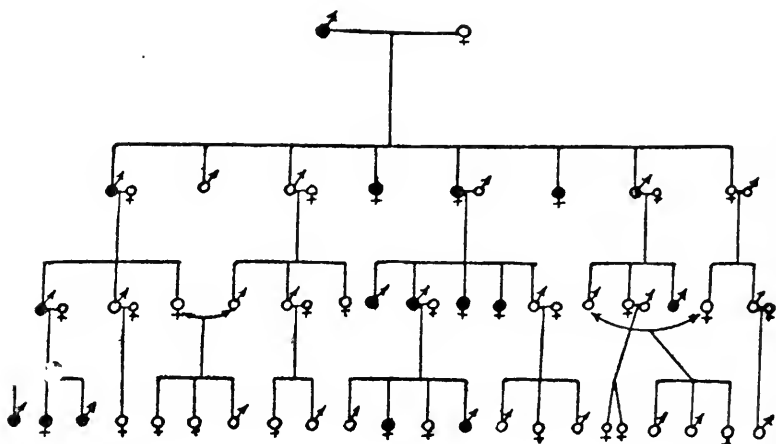


FIG. 2.—Scheme of Dominant Inheritance.

exact photographic measure that one half of the face may correspond to the mother's face whilst the other half corresponds to that of the father.

(c) Considering now the hereditary diseases of the eyes, permit me to mention briefly (more extensively, *vide* my article "Sobre la aplicación del mendelismo en oftalmología," *Anales de la Academia de Ciencias Médicas de la Habana*, January, 1912), that the modern theory of inheritance recognizes three types of hereditary transmission:

1st. Dominant inheritance: characterized in that the stigma, for instance, the disease, is inherited directly from father to son, grandchild, etc., from one generation to the next, attacking in equal number men and women, and in that consanguineous marriage has no importance. The descendants of healthy



individuals do not transmit the disease to their progeny. Figure 2 shows a scheme of the Dominant Inheritance.

2d. Sex-limited inheritance: characterized in that it is inherited only exceptionally in the direct form, from one generation to the next, but almost always skips one generation; in that men are affected ten times more than women; in that healthy men do not transmit the disease, and that sick ones transmit it very rarely; while the daughters transmit the disease as a rule without being affected themselves. Consanguineous marriage has no great importance. The following represents a scheme of sex-limited inheritance:

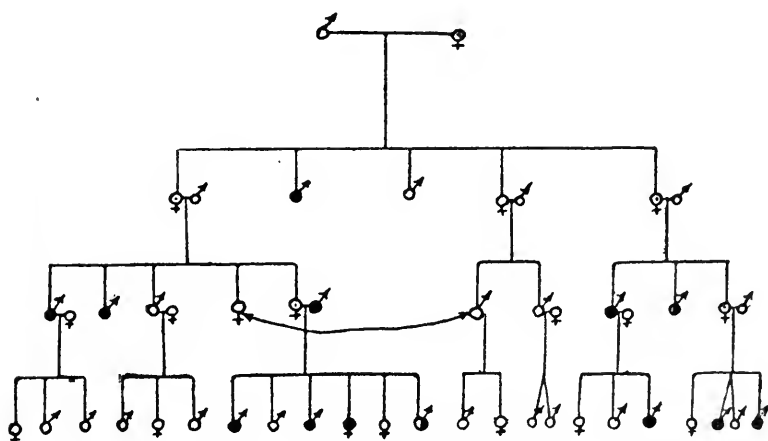


FIG. 3.—Scheme of Sex-Limited Inheritance.

3d. Recessive inheritance: characterized in that in the ascendance, sick individuals are not to be found in important numbers, but in scattered cases, sometimes many generations back; suddenly it reappears in the same family in a large number of persons, attacking indiscriminately men and women. Consanguineous marriage is of the utmost importance, this being in almost twenty-five per cent. of the cases the cause of the reappearance.

It seems difficult to give the proof of the unilateral inheritance in anomalies inherited dominantly, their prototype being cataract, glaucoma, and ptosis, because it is not always easy to mark the line between the hereditary forms and those

occasioned by other agents. But every eye specialist has seen cases in which one eye had been operated upon for cataract at a relatively early age, and the other eye remained exempt from lenticular opacity until a very advanced age. A thorough investigation of parentage will also demonstrate in this many interesting things. We have before all to eliminate the asser-

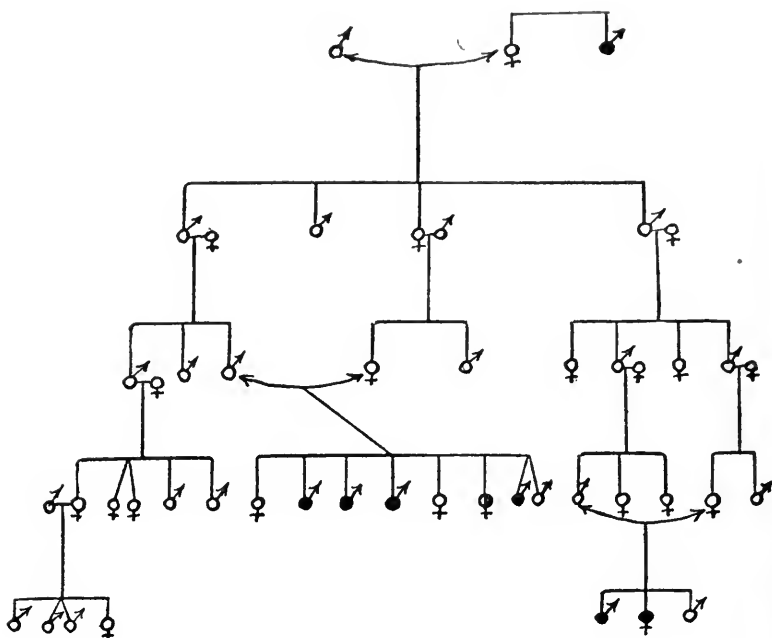


FIG. 4.—Scheme of Recessive Inheritance.

tion which is still to be found in so many manuals, that hereditary diseases of the eyes are always developed bilaterally. To-day we can only say that hereditary disease appears as a rule in both eyes (*nulla regula sine exceptione*).

In the sex-limited inheritance, the development of a unilateral disease has also been proven, as did Kayser with megalocornea (*Klinische Monatsbl. f. Augenheilkunde*, 1914). Another unilateral case of megalocornea combined with heterochromia was described by me in *Zeitschrift f. Augenheilkunde*, 1908.

Flesch observed unilateral megalocornea combined with angioma of the same half of the face (*Wiener mediz. Gesell-*

schaft, 1913). Von Hippel described a case of unilateral Daltonism ("Blindness as to Green and Red Colors") in Graefe's *Archiv f. Ophthalmologie*, xxvi. Other cases have been observed by Kolbe, Edridge-Green (*Lancet*, 1912), Becker (*Centralblatt. f. Augenh.*, 1888), Holmgren (2 cases in *Centralblatt. f. die med. Wissenschaften*, 1880). Hayes observed an unilateral case of Daltonism in a woman (*American Journal of Psychol.*, 1912). An asymmetry of the hereditary transmission of Daltonism has also been observed in the following forms: Reber has published that in twin boys only one was Daltonist (in both eyes). Nettleship (*Transactions Ophth. Society*, iii., 32,) saw twin sisters who were born with only one placenta and who kept their resemblance until an advanced age, and of which only one was Daltonist in both eyes. Vossius recalls, moreover, in his *Textbook of Ophthalmology* that Holmgren had also seen a patient who had on one side a normal eye, and whose other eye was blind for blue and yellow colors; but as we have not yet pedigrees on tritanopia (blindness for blue and yellow colors), such as we have on other dichromats (protanopia and deuteranopia), it is not certain whether this Holmgren case is a unilateral case, acquired or inherited. Vossius further cites Piper's observation of a patient who in one eye was blind for blue and yellow colors (tritanope), and in the other, blind for all colors (monochromate). On hereditary nystagmus which is inherited sometimes in the dominant manner and another time in the sex-limited manner, no unilateral cases are known; but I believe that it will only be a matter of time when we may find them. It will be much more difficult to give clear proof of the unilateral presence of the hereditary optic atrophy, or of the amaurosis familiaria idiomatica.

In the recessive inheritance we have various valuable observations on unilateral existence. Thus, Becker has given the description of a unilateral case of congenital monochromatism ("Congenital Blindness as to All Colors," *Archiv f. Ophthalmologie*, 1879, xxv.). Vossius has described a unilateral case of retinitis punctata albescens. A large number of observations on retinitis pigmentosa unilateralis already exists in the literature of the subject. Gonin as well as Dufour (*Annales d'oculistique*, 1902-1903) and Deutschmann (*Beiträge*

2. *Augenheilkunde*, 1891) have been able to verify anatomically the unilateral existence; other observations come from Dumont, Pedraglia, Ginsberg, Anke, and Rosenbaum; Baumeister has described (Gräfe's *Archiv f. Ophthalm.*, xlx., 1873) a unilateral case of retinitis pigmentosa with deafness on the same side.

It almost seems that the larger number of cases of unilateral existence of a hereditary disease of the eyes is found in the recessive inheritance; but the number of our observations is yet too limited to tell positively in which of the inheritance forms the unilateral transmission is more frequently found.

(d) The unilateral ocular inheritance is proven not only in anatomical and physiological anomalies, but even finds its expression in such refined circumstances as the unilateral immunity. Otherwise we cannot satisfy ourselves as to these cases of unilateral trachoma, which remain during years unilateral, without any measure or precaution having been taken to avoid the infection of the second eye. Axenfeld also observed unilateral immunity in pneumococcus conjunctivitis, and even in serious blennorrhagia. During an influenza epidemic in the Maternity House, which attacked the eyes of nineteen children, I also observed a child aged twelve who had abundant secretion in only one eye for nearly ten days, and whose other eye was not infected notwithstanding that no precautions were taken.

(e) Seeing thus that the correlation of the pair organs may be lost totally or partially, unilaterally by the complicated mechanism of inheritance, we can also search for anisocoria based on unilateral inheritance. Frenkel, Henri ("Études sur l'inégalité pupillaire dans les maladies et chez les personnes saines," *Rev. de méd.*, 1879), divides anisocoria in three groups:

1st. Organic anisocoria occasioned by lesions in the different parts of the pupillary reflex way.

2d. Functional anisocoria which appears only transitorily during diseases such as pneumonia.

3d. Morphological or physiological anisocoria. Thus, it seems to me that we can only accept a physiological anisocoria, when we are able to prove that all parts of the pupillary reflex way are intact; at the present time we do not know how to make this examination with certainty. But if we should succeed in excluding all the disturbances of the pupillary reflex

path caused by diseases or congenital malformations in cases of congenital anisocoria, we should place such anisocoria equal to an anisocoria based on unilateral inheritance. Such an acceptance would be more probable if we should succeed in proving a unilateral inheritance, for instance, of the face. The physiological anisocoria is doubtless a very rare phenomenon. Reche, A. (*Deutsche mediz. Wochenschr.*, 1893) found among 14,992 cases of anisocoria, only 145 without cause, to wit: 1%. The pupillary difference between the diameter of the pupils was 0.5 to 3mm. Reche concludes from his abundant material that anisometropia could not be the cause of physiological anisocoria, the opinion for which Schön and Oppenheim had worked. Körbling demonstrated that the pupillary difference between an average myopic eye and an average hyperopic one was at least 0.5mm. Frenkel proved that an inequality of the visual acuity of both eyes could not produce a permanent anisocoria. From these considerations we deduce that the physiological anisocoria is not yet a well-defined anomaly and that care should be taken in diagnosing morphological anisocoria is seen from the following observations:

CASE I.—P. T. José Maria, 15 years of age, laborer, very healthy, robust, well fed, without the slightest symptoms of neurasthenia or hysteria. He knows nothing about his anisocoria; he comes from a healthy family. He himself was never sick before; nor had he received a traumatism in his head. There are no lymphatic glands in the neck, paresis sympathica, pulmonary disease, dental disease (caries), nor palatal disease. Trigemini sensibility normal. Javal in both eyes: 1°.

Sight: R. E. = refraction + 1.50; L. E. = refraction + 1.50. Dioptric apparatus transparent. Fundi normal. Right iris slightly more clear (brown) than the left one. Anisocoria observed without the slightest change during nine months. There are no traumatic lesions of the sphincter muscle. R. E. : L. E. = 2.50mm : 7mm. Day illumination. R. E. : L. E. = 3.00mm : 6mm. Dark room. Normal reaction in both eyes: to direct and consensual light, to convergence and accommodation, to irritation of the trigemini. Cocaine expands both pupils in the same manner, and in the same time, and first in the inferior section. After twenty minutes the following is observed: R. E. : L. E. = 7.50mm : 8mm.

The sense for colors is normal in both eyes (Nagel's test).

The visual field is normal in both eyes. The stereoscopic sense is perfect; there are simultaneous vision, stereoscopic vision, and perception of relief. There is no diplopia (examination with the red glass). There exists always a tendency to convergent squint, and then pupils show a marked myosis, the right pupil remaining nevertheless narrower.

The ocular movement is somewhat uncertain and tremulous; it is hard for the patient to distinguish objects with a firm sight. The ocular movement may be compared with ataxia in sclerosis multiplex.

CASE 2.—G. M., 34 years of age; married three years; she had two miscarriages, and has two children. She comes from a healthy family. She knows nothing about her anisocoria; she was never sick; she never received a traumatism in her head. There are no lymphatic glands in the neck, pulmonary disease, asymmetry of the face, paresis of the sympathicus, dental disease (caries), nor palatal disease. She has a tendency to obesity. Javal in both eyes: 0.50 D. Sight: R. E. = refraction + 1.00; L. E. = refraction + 1.00. Dioptric apparatus transparent. Fundi normal. Ocular movement normal in all senses; no diplopia. No enophthalmus or ptosis. Pupils: R. E.: L. E. = 6.50mm : 4mm. Day illumination. R. E.: L. E. = 7.00mm : 4.50mm. Dark room. Reaction normal in both eyes: to direct and consensual light, to accommodation and convergence, to irritation of the trigeminus. Pupils not round, but somewhat irregular as in tabes. Cocaine expands both pupils in equal manner and time. Visual field normal. Sense for colors normal. Stereoscopic sense perfect (good perception of relief).

Both of these cases present the type ordinarily called "physiological anisocoria." But upon examining them more exactly the first case shows a certain ataxy of the ocular movement, and the second one, an irregular shape of the pupil, as it often happens in tabes. This proves that the centrifugal part of the reflex pupillary way is not completely normal, and although we cannot say where the lesion is to be found, it does not seem to me fair to consider them as cases of physiological anisocoria based on unilateral inheritance. Doubtless such forms exist, since Frenkel has published some cases, but I believe that they are still much rarer than they are generally considered to be.

3. Ear. It is to Dr. Emilio Martinez, Professor of

Otology in the University of Havana, that I owe the following information on ears, taken from Heimann, Theodor (*l'Oreille et ses maladies*, 1914).

Otology recognizes three different hereditary diseases:

1st. Otosclerosis or ankylosis primaria stapediovestibularis. It is more frequently found in the feminine sex. It is inherited in the dominant manner. The inheritance has been demonstrated by Hammerschlag in four families, by Körner in three. Heimann saw a unilateral case in a physician. The physician's father, as well as two of his sons and one daughter, had the same disease; moreover a sister of the physician, two daughters, and one grandchild suffered from same.

2d. Surditus labyrinthica congenita: or congenital hypoplasia ganglii spiralis. (Alexander has demonstrated the dominant transmission and also a unilateral case.)

3d. Surdi mutitas congenita, or aplasia nerv. cochlearis. The transmission keeps an exquisitely recessive character. In at least twenty-five per cent. consanguineous marriage of the parents is to be found. It is somehow more frequently found in the masculine sex, and it is associated often with retinitis pigmentosa (Schmaltz) or albinism (Dahl). A unilateral case would represent that of Baumeister above mentioned (paragraph "recessive diseases of the eyes").

To these observations of Heimann we add the deafness of white cats with blue eyes, above cited, and whose unilateral forms have been several times observed. Darwin makes mention furthermore of an observation of Sedgwick, that persons who are color-blind from birth sometimes do not perceive musical tunes (*Medico-Chirurg. Rev.*, 1861). As unilateral Daltonism has been proven, we may suspect that there exists also a congenital impossibility to perceive the musical tunes on only one side. This syndrome of Sedgwick (Daltonism plus impossibility of perception of musical tunes) could be called "negative synopsia," in comparison with the positive synopsia (photismo: = induced sensation of color; phonismo: = induced sensation of tune), in which a certain tune awakens the impression of color, or vice versa, and whose hereditary transmission has been proven by Lomer (pedigree of four generations, *Archiv f. Psych. u. Nervenkr.*, xl.).

D. The nervous system contains a series of organic diseases,

whose hereditary character has been universally acknowledged. From these nervous hereditary diseases we must separate the neuropathic inheritance, which is almost always heteromorpha or dissimilar. Raymond says, in his *Études des pathologies nerveuses* (1910), that nervous hereditary diseases are, first, homologous, that is to say, the pathological variation always appears in the same form; they are, furthermore, homochrones, that is, appearing in the same age; and finally, homotopics, that is, the pathological change always appears in the same part of the body. Almost all of these diseases are systematized, but sometimes they affect different systems at the same time. They are abiotrophic diseases, that is to say, occasioned by atrophy of the corresponding cells of the neuron; they never are inflammatory. In the same parentage, these diseases always appear in the same manner, but the same diseases may show different variations. By organic diseases of the nervous system, whose hereditary character has been universally acknowledged, we have:

Tremor hereditarius  
Chorea de Huntington  
Hemicrania  
Dupuytren's disease  
Paramyoclonus multiplex  
Ataxia cerebellaris hereditaria  
Paralysis bulbaris progresiva familiaris  
Ptosis familiaris tardiva (Dutil)  
Infantiler Kern-schwund (Moebius)  
Friedreich's disease  
Paraplegia cereбрalis  
Menzel's disease  
Atrophia spinalis muscularis hereditaria

These diseases have been acknowledged, studied, and separated, only within the last fifty years, and this explains why we do not yet have real pedigrees of them, well studied, as we have, for instance, in ophthalmology. From their complicated character it is easy to understand that the familiar anamnesis cannot help us, therefore we have left only the duty of making exact notations, that our nepotes may profit from them, as did



Keppler from those of his predecessors. As publications on these diseases are very much scattered, it has been impossible for me to find reliable observations on unilateral or asymmetrical inheritance, notwithstanding that the splendid library of Dr. José Valdés Anciano, Professor of Neurology at the University of Havana, was at my disposal, from which I have taken the majority of my notations.

Unilateral cases of ataxia cerebialis hereditaria could be: Turner's case (*Thèse de Paris*, 1856) and that of Cramer (*Beitrag z. pathol. Anatomie*, 1892).

Spiller cites (Osler's *Modern Medicine*) thirteen unilateral cases of sclerosis lateralis amyotrophica, which disease approaches very nearly the hereditary diseases of the nervous system; but these cases were all converted, later, into bilaterals.

E. By unilateral inheritance one could perhaps also explain some cases of dual personality, which is found so often in hysteria and epilepsy. This should then represent the unilateral inheritance of the psyche. Of interest also are the hallucinations. We accept to-day that they are produced by changes in the different afferent sensorial or sensitive ways, which conduce the stimuli to brains of abnormal and especially hereditary conditions, called hallucinogenous conditions. We have many observations which show that these hallucinations are sometimes exteriorized in a constant manner to only one side of the body, to one ear or to one eye (even in a hemiopic form). Wormser has in his thesis (*Sur des hallucinations unilatérales ou dedoublées*, Paris, 1895) recollected twenty-five cases out of the literature. Ziehen refers in his *Physiologische Psychologie*, 1911, a patient who suffered in one ear constantly of hallucinations of a disagreeable character, whilst he had in the other ear only others of agreeable character.

Now, as I already demonstrated in a previous article ("Über einige Stammbäume und die Anwendung der Mendel'schen Regeln auf die Ophthalmologie," von Graefe's *Archiv f. Ophthalmologie*, 1911, Bd. lxxix.) the loss of the hereditary correlation between pair organs cannot be explained in any manner by Mendelian rules which have attained such importance in the inheritance doctrine. Neither can Galton's theory give a sufficient explanation, according to which we

should have a unilateral inheritance only in cases of crossings between two ascendancies, which practically never takes place; and even in such a rare case, we would have an unilateral inheritance only in 20% (*vide* my article *Deutsche Mediz. Wochenschr.*, 1910, No. 24); such explanation would exclude almost certainly the familiar appearance of a unilateral anomaly, as, for instance, heterochromia iridum, which has been observed by Fuchs and de Schweinitz in several members of the same family. The explanation of such phenomenon can only be based on the karyokinesis.

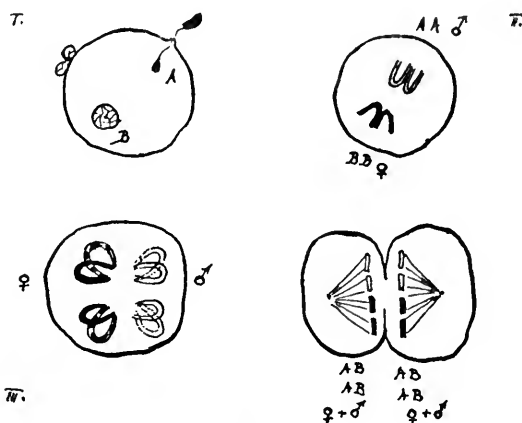
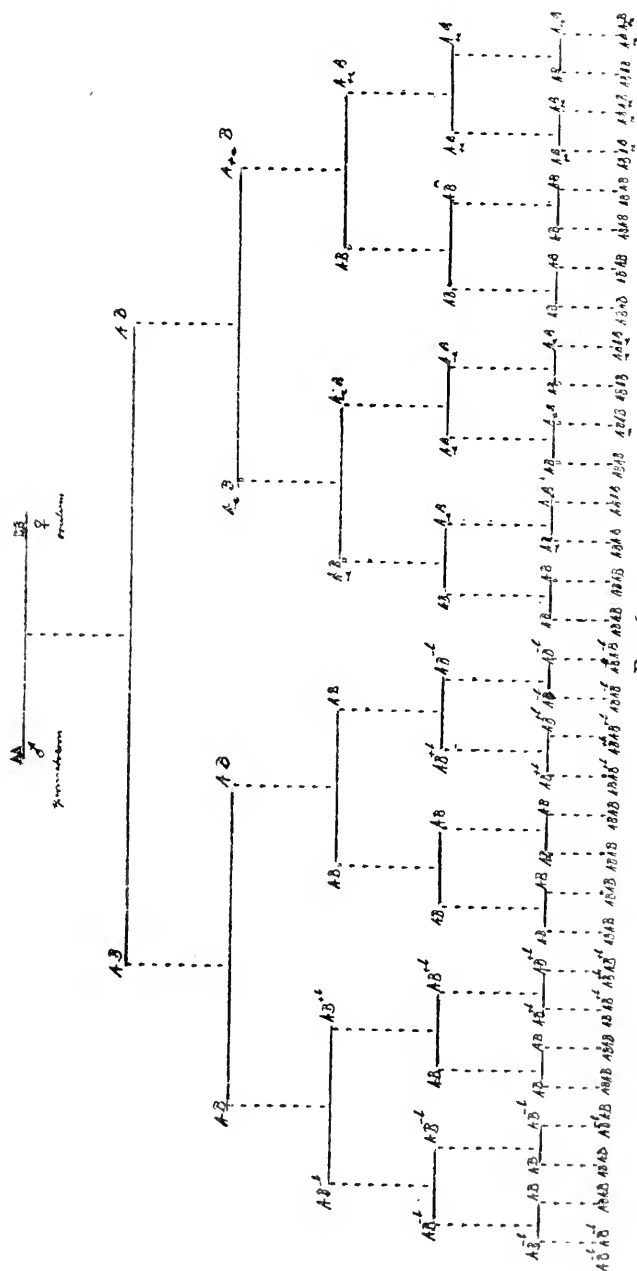


FIG. 5.

According to this theory, the bearers of the hereditary unities are the chromatin corpuscles, distributed in the chromatin loops. After the fecundation of the right ovulum by the spermatozoid, each chromatin loop is divided by the tangent in two equal ones, and the two new cells possess an equal amount of chromatin from the ovulum and from the spermatozoid. Now, by mere calculation of probability, we have to expect, that not always the same amount of AB goes on both sides (in the same manner in which a dexterous sharpshooter places his shots sometimes more to the right side of the center, and at other times more to the left). For this reason we should not be surprised to find sometimes marked asymmetries caused by the ancestral inheritance, but, on the contrary,



that they are not more frequently found. We have again to marvel at the precision of nature's works. These considerations explain also why we find as a rule a slight asymmetry of the face, and why we find complete harmony almost solely in deities' images. Furthermore, it explains to us why twins are so much more alike than brothers who are not twins.

The asymmetrical distribution of the chromatin corpuscles in the unilateral inheritance could be algebraically represented in the form shown in Fig. 6. This algebraical model clearly shows that the body asymmetry will be greater if the unequal distribution of the chromatin corpuscles begins early. This explains in turn why strong asymmetries, such as unilateral albinism, will be found much more rarely than, for instance, a unilateral affection of the eyes.

These demonstrations prove that by studying unilateral anomalies, we may contribute to elucidate the inheritance problems, especially in their mosaic form which Goethe so jovially described in his verses:

“Vom Vater hab ich die Statur,  
des Lebens ernstes Führen,  
Vom Mütterchen die Frohnatur  
und Lust zu fabulieren.  
Urahnfrau liebte Schmuck und Gold,  
das spukt so hin und wieder;  
Der Ahnherr war der Schönsten hold,  
das zuckt noch durch die Glieder.  
Sind nun die Elemente nicht  
aus dem Komplex zu trennen,  
Was ist denn an dem ganzen Wicht  
noch Original zu nennen?”

## ACUTE BILATERAL RETROBULBAR NEURITIS OF SEPTIC ORIGIN, RESULTING IN RAPID AND COMPLETE LOSS OF VISION.<sup>1</sup>

By DR. WALTER EYRE LAMBERT, NEW YORK.

**A**LTHOUGH the eye history of this case, previous to the attack of retrobulbar neuritis, has no special bearing on the subject, it is in itself interesting, and I shall report it briefly.

The patient, Mrs. S., first consulted me in 1896—her age then was 44 years. She is a lady of means, and had of course most comfortable surroundings, but never seemed very well, and had a great many things to worry her. At that time she was complaining of severe headaches, which she referred to her eyes. She had always had trouble with her eyes, and was wearing glasses at the time. The refraction for the right eye was plus 1.25, and for the left eye, minus 1.25. With these glasses she had  $\frac{20}{20}$  vision in each eye. I found a hyperphoria of 3 degrees. At that time I questioned whether this small amount of hyperphoria was the cause of her eye symptoms. My experience since then has shown me that small degrees of hyperphoria frequently produce asthenopia; and correcting it usually gives relief. I prescribed for her glasses with a prism of 2 degrees; these apparently gave her immediate and perfect relief, which continued for several months; when suddenly a pair of new glasses brought on a return of the headaches and feeling of eye strain. She submitted these glasses to me at once; and I found that the optician had placed the prism base up instead of down. The correction of this mistake relieved the patient immediately, and she continued to have comfortable use of her eyes for many years—with some modification of her glasses for increasing presbyopia.

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<sup>1</sup> Read at meeting of American Ophthalmological Society, New London, July, 1918.

On March 27, 1917, the patient consulted me. I had not seen her for over a year, during which time she had been in rather poor health, suffering with severe pains in her head, and neuritis in her neck and arms. She had been under the care of several physicians in Boston; and for some time had been treated by an osteopath there, whom she felt relieved her very much. The eye condition at that time had changed but very little—a slight increase in the amount of hyperphoria, it being 4 degrees. I changed her glasses and advised her to consult her family physician in New York, who had taken care of her off and on for many years. I even then suspected the probability of some sinus trouble. An X-ray was made, the report on which is as follows: "A root fragment had been pushed into the right antrum. The antrum also shows a distinct occlusion of its air space, which possibly may have been caused by the displaced root fragment. The rest of the sinuses appeared quite normal. There is considerable constriction in the nares, caused by the deviated septum and enlarged turbinates. The sella is smaller than the average. There is no evidence of any infection about the teeth." The antrum was operated on April 6, 1917, and found to contain a considerable amount of green pus. Permanent drainage was established through the nasal cavity. The final bacteriological report of cultures from the antrum showed streptococcus viridans. The blood culture proved negative. The patient seemed to be relieved of her headaches, subsequently to this operation; although a slight septic temperature continued. She made an uninterrupted recovery as far as the operation was concerned.

On May 7, 1917, she complained of blurring of vision in the left eye. About this time the headaches had returned with some intensity, and the temperature was a little higher, which was running between 100 and 101. On May 8th, the next morning, I saw her, and found the pupil of the left eye moderately dilated, no reaction to light, and not even light perception. An ophthalmoscopic examination showed a marked, though moderate papillitis—the arteries small, and sluggish, segmented circulation in the veins. The right eye was unaffected, vision being normal. Dr. Weeks saw the patient on the same day, and agreed with me that there was partial occlusion of the central artery of the retina; probably thrombosis of septic origin.

On May 10th, the right eye became affected; a similar ophthalmoscopic picture presented itself in that eye—vision reduced to fingers. Dr. Weeks again saw the patient with me, and we agreed that an immediate operation of the ethmoid cells—which we regarded as the origin of the trouble—should be done. This was in the evening of May 10th;

the operation, however, was deferred, and the next day an X-ray was made, which gave no evidence of sinus involvement.

On May 12th the patient was seen by Dr. Knapp with me: the vision of the right eye had slightly improved, and we were able to demonstrate a distinct central scotoma for form and color, tenderness on pressure on the globe, and also on movement of the eyeball. We regarded her condition as acute retrobulbar neuritis; and the question of an operation on the ethmoid was again considered. But it was determined after very careful consideration, and consultation with two well-known rhinologists, futile. The vision of the right eye failed rapidly, and on May 15th the patient was perfectly blind. Atrophy of both optic nerves followed.

When the case was first seen, both Dr. Weeks and myself were of the opinion that there was a thrombosis of the central artery of the retina—probably of septic origin. After the right eye became involved, and we were able to demonstrate the presence of central scotoma for color and tenderness in the orbit, the diagnosis of an acute retrobulbar neuritis was made—in which diagnosis both Dr. Weeks and Dr. Knapp agreed. Notwithstanding the negative findings of the X-ray examination, and that the ocular complications did not manifest themselves for a month after the operation on the antrum, I am inclined to the opinion that this case is one of focal infection, the focus probably being in the posterior ethmoid cells, extending from the diseased antrum. The fact that the left eye was the first to be affected, whereas it was the right antrum that was diseased, is not uncommon. This contralateral visual disturbance in nasal sinus disease has been frequently observed by Onodi, as is stated in Knapp's *Medical Ophthalmology*. Whether the exploration of the ethmoid cells would have been of any benefit, as the process was so acute and so intense, is, of course, extremely doubtful.

This case illustrates to me a very wise remark our distinguished guest, Sir James McKenzie, made in New York, recently: "Let us not be exclusively Laboratory Physicians."

## CATARACT IN RETINITIS PIGMENTOSA; ITS PATHOLOGY AND TREATMENT.<sup>1</sup>

By DR. ARNOLD KNAPP, NEW YORK.

IT is a well-known clinical fact that a cataract frequently develops in the course of retinitis pigmentosa. Thus Leber<sup>2</sup> states that this cataract is so constant in the older cases that it often suggests a diagnosis of the condition. The cataract develops in the form of a star-shaped opacity at the posterior pole, limited to the posterior cortical layers. It remains limited to these layers for a long time and rarely develops into a total cataract. In the beginning there is a punctate opacity at the region of the posterior pole, to which one or more radiating opacities are joined, suggesting a star shape. These radiating opacities render the examination of the eye-ground difficult. This cataract is rarely accompanied by other forms of cataract formation; an associated opacity in the anterior layers has been described, and I have observed a well-defined rounded opacity just anterior to the nucleus in two cases of posterior polar cataract in retinitis pigmentosa.

This posterior polar cataract, though it advances but very slowly, seems to be greatly responsible for the reduced central vision of these patients. Doyne<sup>3</sup> was struck by the remarkable effect produced by the removal of the lens in retinitis pigmentosa, even when the posterior capsule is alone affected and not to a very great degree. Though it is generally believed that the prospect of sight after the removal of the lens is poor on account of the nature of the disease, Doyne states that this

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<sup>1</sup> Read at meeting of American Ophthalmological Society, New London, July, 1918.

<sup>2</sup> Graefe-Saemisch, *Handbuch*, vol. vii., pt. ii.

<sup>3</sup> *Ophthalmoscope*, 1910.



is directly opposed to the facts. The improvement that occurs after the removal of the lens, even when so little affected that the normal observer can see every detail of the fundus without difficulty, this author regards as nothing short of marvelous. Doyne has performed the operation several times, and with one exception,—in which central vision was grossly affected,—he was struck by the improvement, which was out of all proportion to his expectations. His experience in this class of cases is so positive that he urges most strongly the removal of the lens in the presence of a posterior capsule that makes a difference in the examination of the eye.

After my attention was drawn by Dr. E. J. Curran, now of Kansas City, to Doyne's views on the advisability of extracting immature cataracts in retinitis pigmentosa, I have been on the lookout for these cases, and I now wish to report on four cases which have been operated upon by me.

CASE 1.—C. C., aged 65, was seen June 8, 1914, because the left eye had been failing for four years. Sight in the right eye was  $\frac{3}{4}\%$ ; field was concentrically contracted to from  $2^{\circ}$  to  $5^{\circ}$  from the center. There was the usual posterior polar cataract in the left eye, together with a small opacity apparently on the anterior surface of the nucleus of that lens. The eye-ground presented the usual changes. Wassermann negative. The cataract was removed in the capsule without incident. After operation the vision with correcting glass was  $\frac{7}{8}\%$ . There were some opacities of the vitreous and an opacity on the hyaloid from a hemorrhage occurring at operation. Subsequent vision was probably better as soon as the opacity was absorbed, but it has been impossible to reëxamine the patient.

CASE 2.—M. S., aged 29, has never had good sight, especially for the last four years. Vision R.:  $\frac{2}{3}\%$ , L.:  $\frac{1}{4}\%$ . Field contracted to  $5^{\circ}$  about center, typical radiating posterior cortical opacity in the lens. The eye-ground presented the usual picture. Extraction with capsulotomy was performed, without accident, on the right eye; after a few days a severe iridocyclitis developed which resisted the usual methods of treatment and did not subside until three abscessed teeth in the upper jaw on the same side were extracted. The resulting thickened secondary cataract was operated upon about eight months later; vision at present with glass is  $\frac{2}{3}\%$ .

CASE 3.—I. S., aged 32, always suffered from night-blindness; the sight in the daytime has been failing for four

years. Vision is  $\frac{3}{20}$  in each eye with the usual posterior cortical cataract and the fundus changes of retinitis pigmentosa. Combined extraction with capsulotomy, no reaction, followed by needling with a resulting vision of  $\frac{1}{20}$ . A thin after-cataract has re-formed, which, after another needling, should give still better vision.

CASE 4.—S. P., aged 52, has been troubled with night-blindness for many years. Gradual diminution of sight in the daytime. Vision equals  $\frac{3}{20}$ . Posterior polar cataract with advanced changes in the eye-grounds. Field very narrow. The right cataract was extracted in the capsule without event. The result of the operation was excellent, with perfectly clear media. With a correcting glass vision was only  $\frac{6}{20}$ . There was no optical impediment nor particular macular disturbance to explain this poor sight, except that the field was very small and the patient's mentality unusually dull.

To recapitulate, in these four cases—two of which were operated on by removing the lens in the capsule, and two with capsulotomy—the visual results were: 1,  $\frac{2}{20}$  improved to  $\frac{2}{20}$ ; 2,  $\frac{2}{20}$  improved to  $\frac{3}{20}$ ; 3,  $\frac{3}{20}$  improved to  $\frac{1}{20}$ ; 4,  $\frac{3}{20}$  improved to  $\frac{6}{20}$ .

The method of operating required in these cases is not different from that of the usual cataract. The operation is not any more difficult. Doyne observed in every case that he had considerable difficulty in getting the lens away after opening the capsule, as if it seemed to be unusually adherent. I was not able to confirm this peculiarity in the delivery of the lens. After-healing is uneventful. There are no complications as far as the original eye condition is concerned. Doyne has observed that after extracting the lens the capsule has a tendency to form again; a film recurs in the pupil exactly like a wrinkled capsule that requires needling. The visual results are distinctly encouraging and I entirely agree with Doyne on the correctness of removing the lens under these conditions.

*Pathology.*—One of the cataracts extracted in the capsule was prepared for microscopic examination with the following findings. The capsular epithelium at the equator of the lens is wavy in outline, the cells are enlarged and have lost their outline; their protoplasm is granular. Underneath this layer there are irregular vacuoles and the adjoining nucleated lens fibers present vacuoles and are generally irregularly distributed.

At the epithelial boundary the new formation of lens fibers has taken place irregularly and to an increased degree. A thin band of elongated epithelial cells continues along the posterior capsule. Directly internal to this are nucleated cells which are vacuolated, distended, and have proliferated irregularly, often forming nucleated lens fibers. Just at the center and occasionally above and below the center there are irregular cavities filled with poorly staining lens detritus, albuminous material, globular bodies, and the edges of the bordering lens fibers are corroded; these cavities represent the lenticular opacities seen with the ophthalmoscope.

The changes therefore found in this form of cataract are changes in the equatorial capsular epithelium, prolongation of transformed capsular epithelium along the posterior surface, and irregular cavities of lens detritus in the posterior cortical layers.

The literature contains but few accounts of microscopic examinations of cataracts occurring in retinitis pigmentosa. The best description is that given by Wagenmann,<sup>1</sup> in which similar changes to those found in the above-described specimen are given.

<sup>1</sup> Wagenmann, "Pathologische Anatomie d. Retinitis pigmentosa," *Arch. f. Ophth.*, vol. xxxvii., 1891.

ANNULAR LENS OPACITY FOLLOWING CONTUSIO  
BULBI. THE REPORT OF A CASE AND A  
THEORY RELATIVE TO ITS FORMATION.

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(With one illustration on Text-Plate XXI.)

VOSSIUS (1), in a session of the Medical Society of Giessen, 1903, was the first to call attention to an annular lens opacity occurring after ocular contusion. Three years later Keller (2) reported six cases and accurately described the lesion. Since that time, according to Casey Wood (3), some thirty or more cases of traumatic ring-shaped cataract have been recorded in the literature.

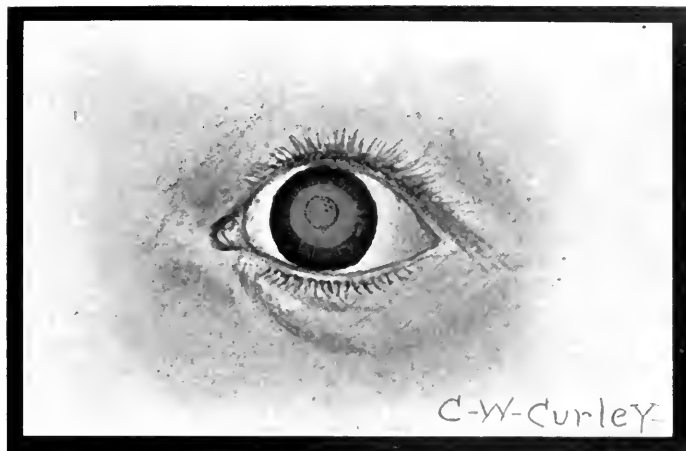
The following case not only presented clinically an annular lens opacity, but exhibited also a "macular hole," a lesion not so common as to render its clinical report *mal à propos*.

CASE REPORT

John S., an apprentice in an automobile shop, while assisting in the lighting of a welding apparatus, was struck on the left eye by a piece of hose, following an explosion.

The patient presented himself for examination January 4th, one hour after the accident. The left eye exhibited an excoriation of the skin of the lower lid, a small superficial laceration of the tarsus of the lower lid beginning at the free border of the latter, between its outer and middle third, and extending obliquely outwards for a distance of 7mm towards the retrotarsal fold, and a small laceration of the ocular conjunctiva between the limbus and the external canthus, with ecchymosis in and about the lesion; marked ciliary injection, cornea clear, aqueous turbid, vision of the right eye  $\frac{2}{20}$ , left eye  $\frac{2}{20}$ , pupils equal. Because of the

TO ILLUSTRATE DR. BERRISFORD'S ARTICLE ON "ANNULAR LENS  
OPACITY FOLLOWING CONTUSIO BULBI."





marked blepharospasm and general discomfort of the patient further examination was not attempted.

On January 6th, the patient complained only of poor vision in the left eye. Oblique focal illumination and the loupe disclosed no lenticular abnormality. By transmitted light could be seen a circular circumscribed lens opacity having a diameter of 3mm, its center being situated slightly nasal to the corneal center, on or directly beneath the capsule. This annular opacity appeared to be made up of minute dust-like dots, some of which coalesced to form small masses. These masses seemed translucent, lardaceous in structure, and brownish in tone. This discrete ring appeared heavier and slightly broader in the superior temporal portion, least distinct in the nasally directed portion of the circle, being at no place broader than a hair. The area enclosed within the ring appeared clear excepting for a few scattered dust-like opacities situated chiefly in the superior temporal quadrant.

The fundus showed in exactly the macular region, a sharply circumscribed oval area occupying a transverse position, with a long diameter approximately one third that of the disk. This area contrasted strongly with the neighboring fundus, being sharply delimited, dusky red in color, and appeared to contain within it minute pigment deposits, as if the area were dusted with emery powder. At this time parallax displacement disclosed no evidences of a surrounding elevated zone.

January 18th, the patient having resumed his duties, complained that he had difficulty in judging distances. Hering's test and bar reading substantiated his claims. Perimetric findings gave no accurate information regarding central scotoma. The vision of left eye  $\frac{2}{60}$ , with eccentric fixation  $\frac{2}{60}$ . The patient, refracted under atropin, gave the following results: O. D. + .50 S. + .50 C. Ax. 90 =  $\frac{2}{60}$ ; O. S. + .75 S. + .50 C. Ax. 90 =  $\frac{2}{60}$ .

During the following weeks the patient was examined from time to time, the annular lens opacity appearing less distinct each visit, the nasal half disappearing first, later the temporal half, and finally the dots that were within the circle. The annular opacity completely vanished in seven weeks.

Twenty days after the injury the macular lesion presented the typical appearance of a "macular hole," a dusky red punched-out excavation surrounded by an oedematous halo. Later two grayish atrophic patches developed in the red depressed area which in the course of time increased in size. The "macular hole" was observed to change its contour and instead of remaining transversely oval, now appeared roundish by the extension of its superior boundary.

It was perhaps Knapp (4) in 1869 who first recorded a case of "macular hole." Since that time much attention has been given to the study of the macula following contusions of the globe, so that this condition now holds a well recognized place in ophthalmoscopy, its appearance being so striking that if once observed it will scarcely be overlooked.

#### ANNULAR LENS OPACITY.

Traumatic ocular injuries giving rise to an annular opacity of the lens may be accompanied by the following complications: Laceration of the lids, proptosis, extra-ocular palsies, subconjunctival hemorrhages, conjunctivitis, superficial and deep opacities, erosions and perforations of the cornea, perforation and rupture of the sclera, prolapse of the vitreous, mydriasis, myosis, hyphæmia, gelatinous and fibrinous exudation into the anterior chamber, anterior and posterior synechiæ, ophthalmoplegia interna, rupture of the sphincter pupillæ, traumatic cataract, hemorrhage of the vitreous, commotio retinæ, retinal hemorrhages, optic neuritis, rupture of the chorioid, extensive pigment changes in the fundus as well as atrophic changes and "hole" in the macula.

The reported case was complicated by a laceration of the lower lid margin, laceration and ecchymosis of the ocular conjunctiva between the external limbus and the outer canthus, and a "macular hole."

The majority of cases thus far reported are the result of blunt force received from flying objects such as dirt, B. B. shot, footballs, broken drill punches, rocks, etc. However, Gifford (5), Casper (6), Natanson (7), Purtscher (8), (three cases), Holloway (9), Strader (10) have recorded instances due to perforating wounds all of which lay anterior to the lens with the exception of one case recorded by Purtscher (8). That this impact of the foreign body need not have its site on the anterior portion of the globe is illustrated by Steiner's (11) case. A typical Vossius's ring was observed after a shot in the temporal region, the bullet having passed into the orbit behind the eye, the latter remaining uninjured although displaced suddenly forward from behind.

Owing to the delicacy of outline of Vossius's ring, the len-



ticular surface requires a most careful inspection for its disclosure. There can be no doubt that many cases have been either overlooked, unrecognized, or not reported. The pupil should be dilated. Inspection by means of oblique illumination and the loupe is as a rule not sufficient. The author was unable to discover the opacity by this means. The binocular loupe and corneal microscope lend considerable assistance (Nicolai) (12). By means of the ophthalmoscope and the large concave mirror, at a distance of about 33cm, from the patient, the annular lens opacity could be plainly seen contrasting as it did with the red reflex of the fundus. Details were ascertained by using a small concave mirror and a + 20 lens.

The form of the annular opacity as the name implies is usually circular. Rarely is it oval in contour as illustrated in a case of Casper's (6) following a perforation of the cornea with a resulting prolapse of the iris, the ring being drawn out into a point downwards and laterally, corresponding to the defect of the iris. Höeg (13) has reported a case in which the circle was interrupted by clear lines. The lenticular opacity was bordered by a rounded corner corresponding to the upper former site of the temporal pupillary margin, consisting of an arc, the upper portion of which was slightly convex, almost horizontal, and a temporal portion, also slightly convex, but almost vertical. The breadth of the ring is variable. In some cases it is as thin as a hair, in others much broader. The annular opacity in an individual case not infrequently shows a lack of uniformity in both density and breadth, often appearing broader and heavier in its temporal half, the nasal portion appearing so faint as to be almost imperceptible. Such a clinical picture was to be observed in the reported case.

Close inspection in most cases shows the circular opacity to be made up of fine dots crowded, as a rule, closely together and in some instances accompanied by faint lines. These dots, it would appear, frequently coalesce to form small masses, the dotted appearance occurring as a rule in the faintest portion of the circular opacity. The circle is not always complete, occasionally the opacity being interrupted by clear spaces. In some cases the ring is pigmented as the reports of Vossius (1), Nicolai (12), Von Mertz (14), Löhlein (15), Robinson (16), Cates (17), and others will show. Pigmentation need not be

confined to the ring, but may appear outside (Robinson (16), Natanson (7) ), or inside (Strader (10), Cates (17), Gifford (5) ), the area enclosed by the circle. The case herein reported exhibited a few scattered dots situated in the upper temporal quadrant of the enclosure.

Löhlein (15) reported a case which on being viewed with a corneal microscope, showed a circle consisting of radially arranged dots and lines of various sizes extending inwards towards the center of the circle, giving the impression of an opaque disk with a distinct outline.

Hescheler (18) observed a cone-shaped opacity extending downwards from the circle extending to the periphery of the lens, and towards the upper nasal side another opacity the size of a pinhead.

Höeg (13) noted in one case a scarcely perceptible diffuse opacity within the ring, while towards the lower periphery of the lens there were several radial opacities which disappeared in a few days.

The transient character of Vossius's ring is a prominent feature, the opacity completely disappearing in from a few days to eight weeks. The reported case had a duration of seven weeks. The annular lens opacity does not impair vision to any marked degree although the complications of contusio bulbi which frequently accompany it often produce the opposite effect.

The mode of formation of annular lens opacity has been the subject for much discussion, Vossius (19) giving the first explanation: "By the indentation of the cornea following trauma, the iris is forced against the lens producing either an expression of pigment from the cells of the pupillary margin, which are fixed by fibrin on the anterior capsule of the lens, or as in the case of an unpigmented gray ring, the indented cornea forces the pupillary border of the iris against the anterior capsule of the lens producing a degeneration of the capsular epithelium at the point of contact." He bases his last assumption upon the researches of Schirmer (20) who produced experimentally a contusion cataract by the traumatization of the capsular epithelium, which after a time regenerated leaving no visible signs of injury. Through a small peripheral section of the cornea, Schirmer introduced a fine

blunt-pointed probe up to the opposite pupillary border withdrawing it in such a manner that the instrument passed gently onto the anterior surface of the lens. He regularly found a linear, occasionally a longitudinally oval opacity with vaguely defined borders, at the point of injury. When viewed with the loupe, it appeared to have a grayish color and situated beneath the anterior capsule. Occasionally the opacity was so faint that it was hardly perceptible, but when viewed by means of transmitted light was nearly transparent, and of a granular blackish appearance. The opacity appeared from  $1\frac{1}{2}$  to 12 or more hours following operation, its greatest intensity being observed after from thirty-six to forty-eight hours, when it began to decrease, disappearing in from a few days to a few weeks.

A clinical case which would appear to confirm the hypothesis that the cornea is brought in direct contact with the lens in the production of annular lens opacity, is one reported by Krusius (21). He found a faint gray opacity on the posterior corneal surface or on the corneal side of Descemet's membrane at a point corresponding to the opacity in the lens.

The theory advanced by Vossius, substantiated it would seem by the experimental evidence of Schirmer, might at first appear authentic. Nevertheless, clinical evidence renders the supposition untenable for many of the cases at least. To come straight to the point, the indentation of the cornea is not a requisite in the production of annular lens opacity. Small perforating injuries either anterior or posterior to the lens are known to have produced it as the cases of Gifford (5), Purtscher (8), etc., record. The site of traumatization need not necessarily be situated in the anterior segment of the globe as Strader's (10) case gives proof. A bullet lodged deep in the orbit pushing forward the bulb. Höeg (13) affirms that the displacement of the aqueous necessary to produce such a trauma that would bring the cornea and lens in contact over an area 3mm in diameter, could not possibly occur without rupture of the iris or cornea. It is his opinion that a small body applied with force to the center of the cornea would unquestionably result in a lenticular opacity more dense in the center than at the periphery. Certainly this is untrue, for often neither cornea center nor iris is ruptured, the bodies applying

the force varying from B. B. shot and steel chips to saddle horses, footballs, and missiles of clay.

Höeg (13) has advanced a second hypothesis as the causative factor in the production of annular lens capacity, which seems more plausible than that offered by Vossius. It is his belief that the ring is produced by a sudden rise of hydraulic pressure in the anterior chamber to such a degree that the iris is pressed forcibly against the lens. This theory would not be inconsistent with applied force from foreign bodies of various sizes and shapes striking at all angles.

Purtscher (8), Holloway (9), and Steiner (11) aver that in cases where pressure is applied posterior to the lens, the increased pressure in the vitreous drives the lens against the iris producing the annular opacity. Coats (22) refutes this assumption, stating that in his belief the production of the ring from pressure exerted behind the lens is quite impossible unless the iris becomes imprisoned between the lens and the cornea, for the iris will give away before the advancing lens.

Gifford (5) who at first accepted Vossius's explanation for the phenomenon later abandoned it. There came under his observation a patient who received a non-perforating wound of the inner limbus and adjoining conjunctiva of the right eye with a resulting annular opacity. "Here was a typical case of Vossius's ring resulting from a glancing blow of a small piece of copper at the inner periphery of the anterior chamber; an injury which could not possibly have pressed the center of the cornea against the iris." Gifford therefore adopted the more convincing hypothesis of Höeg.

Can we, through the theory of Höeg explain the presence of pigment granules within the ring as is not infrequently the case? This is the *crux criticorum*. The author believes this is the weak point in Höeg's assumption.

In the discussion of the *causa causarum*, operating in the production of this clinical entity the writer will confine himself to the lesion described as consisting of a ring-shaped lenticular opacity about 3mm in diameter composed of pigmented or non-pigmented dots the area within the confines of the ring or outside of it, in certain cases containing like dots. That transient opacities of the lens such as radial lines, streaks, stars, disk-shaped nebulae may occur following contusions of

the globe is well known and has been observed in the presence of the above described lesion. It may be said with a fair degree of certainty that such opacities lie in the capsular epithelium or in the anterior cortex except in rare instances. On the contrary, pigmented opacities such as comprise Vossius's ring or areas adjacent to it can with consistency be said to lie upon the anterior capsule of the lens. If the above statements be true can we preclude the possibility that different pathological processes may be operating in each? The author believes we cannot.

Direct force applied to the lenticular surface after the method of Schirmer (20) or indirect violence brought about by beating the eye for several minutes (Roemer (23) ) can produce experimentally these lenticular radii, nebulae, etc. As far as the writer can ascertain, experimental evidence is wholly lacking to prove that such applied force mechanically results in an annular lens opacity and not the vascular phenomena that accompanies it.

It is the author's belief that a correct explanation of the pigment dots found within the area encircled by the ring is the problem which solves, in part at least, the question of formation of Vossius's opacity.

The plausibility of the assumption that by the increase of hydraulic pressure within the anterior chamber pigment is pressed from the iris and deposited upon the anterior capsule of the lens in the pupillary area must, in the opinion of the author, be received with doubt.

Gifford (5) holds that the disturbance of nutrition which the ring itself might produce may cause the dust-like opacities within the ring. Cates (17) logically contends that if the opacification of the tissue within the ring be due to a disturbance of nutrition produced by the ring, such an opacity would more likely appear of a uniform nature and not granular in character. He adds: "If we accept the premise that the ring, when presenting a brownish color contains pigment squeezed from the pigment epithelium of the iris, may we not, with reason attribute the occurrence of opacities of that color encircled by the ring to a simple migration of pigment particles from a like deposit in the ring itself?"

The theories of Vossius and Höeg rest purely upon a mechani-

cal basis. In the formation of annular lens opacity may not another important factor enter into the discussion? In reference to non-perforating ocular injuries Parsons states: "The typical signs of cyclitis, more especially the presence of precipitates on the back of the cornea are seldom seen after contusions. Just as traumatic iritis in a greater or less degree follows such injuries, there can be no doubt that the ciliary body is also frequently involved. The haziness brought about by exudates in the aqueous must be attributed to iridocyclitis and there is little doubt that the vessels of the ciliary processes often suffer severely. It must be remembered that precipitates on the cornea are evidence usually of a subacute or chronic process, and this is generally absent."

Have we experimental evidence to warrant the above assertions? The pathological sequelæ of applied force to the eyeball is most beautifully exemplified by Bach's (24) work. He traumatized the eyeballs of rabbits with a lever apparatus producing a cloudy transudation in the anterior chamber with myosis ten minutes after the injury. The aqueous cleared and the pupil dilated after three quarters of an hour. Pathologically the anterior chamber was filled with a partly granular, partly filamentous contents, but only on the anterior surface of the iris and in the pupillary area, never at the region of the ciliary processes. Bach believes the cause of the transudation after traumatism is an alteration of the vessels of the iris, a paralysis, the consequence of contusion [which led to extravasation of the albuminous substances of the blood. Microscopically he found the vessels of the iris very much distended and the iris tissue edematous.

It would, therefore, appear that after traumatic injuries of the eye of sufficient severity to produce an annular opacity, an iridocyclitis immediately follows, attested clinically by ciliary injection, myosis, cloudy aqueous, etc. Clinical and experimental evidence shows this in most cases to be of a transient nature, disappearing in a few hours at most.

That the dots comprising the ring and those within it in the observed case appeared in transmitted light, translucent, composed of pigment fibrin and not unlike the minutest K. P., formed in the mind of the writer a basis for a new theory. The deep ciliary injection and turbidity of the aqueous, both

of which were of short duration, led him to believe that the formation of annular lens opacity was of an inflammatory nature, a consequence of traumatic iridocyclitis.

The pathological picture presented by the anterior capsule of the lens may have had its origin in the following manner: As a consequence of the ocular contusion, a mild iridocyclitis results, an exudate from the vessels of the iris and ciliary body pouring out into the aqueous. As a consequence, the fibrin therein contained agglutinates the pupillary border of the iris to the anterior capsule of the lens. From the experimental evidence of Bach it is fair to assume that an attenuation of arrestment of the mobility of the iris of a temporary nature immediately follows the injury. When the iris regains its normal function, an action which takes place after a brief period of time, it frees itself from the fibrinous adhesion at its pupillary border, the fibrinous ring remaining at the point of contact.

How then are we to account for the pigmentation of the ring and the area enclosed by it? The coloration of the circular opacity may be derived from the pigment epithelium of the iris at its point of contact with the lens capsule or may arise from the following sources: uveal pigment contained within the leucocytes that have poured out into the aqueous or pigment derived from the destruction of red blood corpuscles.

The pigment within the area enclosed by the circle or beyond it may be the result of leucocytic migration from the ring itself as Cates (17) suggests, the deposition of pigment-containing leucocytes from the aqueous or pigment derived from the destruction of red blood corpuscles fixed by fibrin. The presence of macroscopical blood in the anterior chamber associated clinically with Vossius's ring is so frequently observed that the author believes this means should not be completely ignored as a possible etiological factor.

In conclusion the author wishes to state that for a more complete elucidation of this most interesting subject the following experimental studies should be attempted: 1. Striking various areas of the cornea and sclera by means of foreign bodies of divers sizes and shapes flying at different angles. 2. Notation of the intraocular pressure at the moment of contact. 3. The recording of microscopically obtained data showing clinically a typical Vossius's ring.

Until this is done we must content ourselves with the several theories advanced accepting tentatively the one which from a clinical aspect seems the most logical.

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## CONCERNING THE ETIOLOGY OF HEREDITARY OPTIC NERVE ATROPHY. REPORT OF TWO CASES WITH INTERESTING X-RAY FINDINGS.<sup>1</sup>

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*(With two figures in the text and six on Text-Plate XXII.)*

**I**N this paper we are concerned only with the etiology of hereditary optic nerve atrophy. The cases here reported present nothing that is new in the symptomatology of the disease, and, as this phase of the subject is thoroughly digested in the monumental work of Wilbrand and Saenger, no essential purpose would be served by restating it.

The opportunity was recently afforded to make an X-ray study of the several members of two families in which the disease had made its appearance, and the results of this investigation are the feature of this communication. The findings are in no way conclusive but are sufficiently uniform to merit attention.

In the first family (C.) there were four children, three boys and one girl. One boy died of typhoid fever, the other two are affected. The girl is living and unaffected.

CASE I.—(J. C.), the first male, who was a stove molder, was affected when 30 years of age. He had the usual children's diseases but suffered no permanent sequelæ and has been in good health since. Smokes moderately and uses but little alcohol. In February, 1912, he accidentally discovered that the vision of the left eye was poor although he could see but large objects. The vision of the right eye was seemingly unaffected. By the latter part of August of the same year he had to give up work because of the poor vision in the left eye. Soon after this a blind spot appeared to the

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<sup>1</sup> Read at meeting of the American Ophthalmological Society, New London, Conn., July, 1918.

outer side of fixation and then spread to the center. He was much annoyed by a continued shower of snow flakes which was finally replaced by a fog. Never had diplopia. When examined in November, 1917, eccentric V. R. E. =  $1\frac{1}{10}$ ; L. E. =  $2\frac{1}{4}$ . Iridic reflexes sluggish. There was an absolute central scotoma about  $20^\circ$  in extent. The optic nerves were greenish gray and the vessels contracted. The neurologic examination was made by Dr. W. G. Spiller and was negative except that the tendon jerks were over-prompt.

The nasal examination made by Dr. J. G. Schwerin showed; nasal cavities negative, tonsils and pharynx red and swollen. The X-ray examination made by Dr. Henry K. Pancoast showed an enlarged pituitary fossa both by deepening and in the anteroposterior direction, 13mm anteroposterior by 12mm deep.

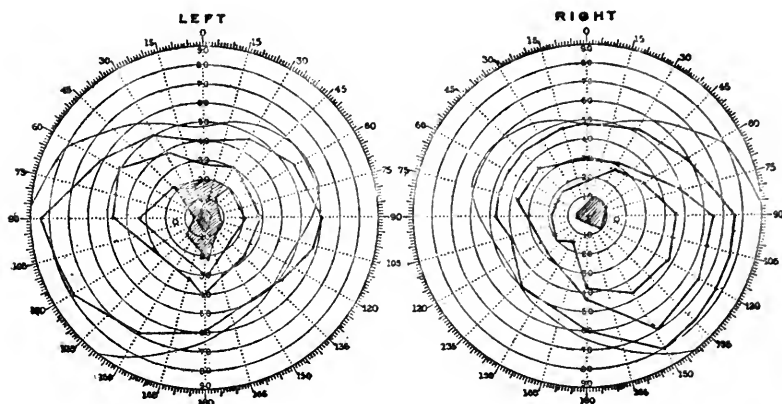


FIG. 1.—Case 2. Fields for form, blue and red. R. E., relative and absolute scotomata. L. E., relative scotomata.

CASE 2.—(C. C.), the second male affected, first seen in October, 1917, was 29 years of age. Until the summer of 1917 he had been employed in a glass works, the last seven years as a blower. During the summer, three weeks previous to coming to Wills Hospital for failing vision, he did outdoor iron painting. The paint contained Japan dryer. He had, however, no toxic symptoms. Three weeks after quitting this work he noticed that persons seen across a street looked blurred. Vision continued to fail until by the following week he could no longer read ordinary print. He thinks the sight of the left eye first began to fail, but of this he is not certain. He was in good health until the spring of 1917, when he had congestion of the lung with pleurisy.

He has smoked about 1 oz. of tobacco a day for several years. Does not use alcohol. He is married and has one child, living and well. His build is slight and he appears anæmic.

Pupils 4mm. Iridic reaction normal. Interpupillary distance 71mm. The upper nasal borders of the papilla are obscured and prominent. V. R. E. =  $\frac{2}{3}\%$ ; L. E. =  $\frac{1}{16}\%$ . Visual field for R. E. shows a scotoma for red extending about 8° on either side of fixation and 20° below and breaking through the limit for red above. At fixation there is an absolute scotoma about 5° in extent.

In the field of the L. E. there is a relative central scotoma of about 5° extent. The form fields showed only slight contraction. It was thought that the condition might be due to an intranasal lesion and this was apparently confirmed by the examination made by Dr. J. G. Schwerin, who found both nasal cavities full of polypi and a purulent ethmoiditis. Complete eradication of these conditions, however, failed to arrest the progressive loss of vision but the œdema of the papilla disappeared. This was followed by an increasing atrophy.

A further search for the cause brought out the fact that a brother had lost his sight from optic atrophy (Case 1). The neurologic examination was negative. The aural examination by Dr. Lewis Fisher showed a hyperactive response in nyctagmus and diminished responses to vertigo and past pointing, indicating a central disturbance. The X-ray examination showed possibly a slight deepening of the pituitary fossa—on the border line of normal size, anteroposterior 10mm; depth 11mm.

The second family (S.) in which this disease appeared consisted of nine children, five males and four females. Of these the fifth and seventh child, both males, are definitely affected, the third, a female, presents suggestive incipient symptoms of pituitary disease. The mother of this family was an only child. She is living at the age of 53 years. The father is 53 years of age and has normal eyes. There is no consanguinity in the ancestry so far as could be determined.

CASE 3.—The first member affected (F. S.) is a male, now 26 years of age. At the age of 12 years the sight began to fail, and in six months' time he was no longer able to read. He thinks the left eye failed the faster. He was examined early in the attack by a competent ophthalmologist when a

diagnosis of optic atrophy was made. Vision has remained stationary in the last thirteen years. Except for scarlet fever at seven he has had no illness. At school he was a normal pupil until his sight failed.

Examined February, 1918; eccentric V. R. E. =  $\frac{1}{120}$ ; L. E. =  $\frac{1}{20}$ , pupils equal, consensually right = 5.5mm, left, 5mm; R. E. slightly divergent. The face is asymmetrical, the right side recedes. The skull is high and narrow. The field of the right is contracted to within 40° of fixation, and there is a central absolute scotoma 20° in extent; the left is moderately contracted, and there is a central triangular absolute scotoma 60° x 15°. No color perception. Both

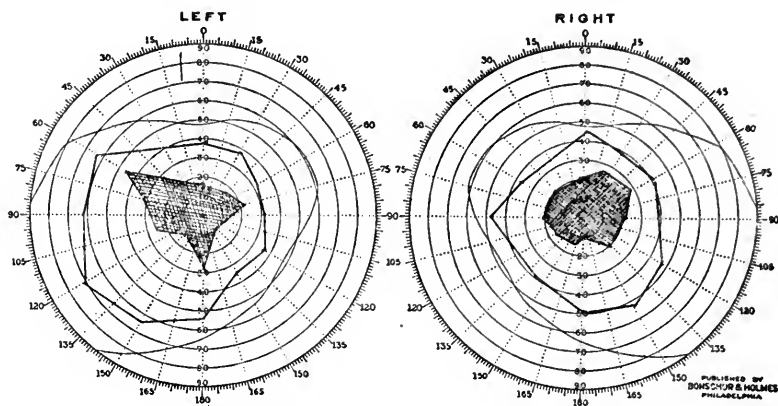


FIG. 2.—Case 3. Form field. Absolute scotoma.

optic papilla are atrophic with sharply defined laminae. The vessels are of normal size in the right and slightly contracted in the left. The arteries are too bright. Under ophthalmoscopic examination the eyeballs develop a jerky movement usually up and to the right.

The physical and serologic examinations are negative. The X-ray examination shows a large pituitary fossa on the border line of normal—12mm anteroposterior diameter and 10mm deep.

CASE 4.—The second male affected (E. S.) is 22 years of age, a chocolate maker for the past nine years. Two years ago he first noticed that colors did not appear normal, and examination of the eyes at that time is said to have shown an inflammation of the optic nerves. One year later he had repeated Wassermann tests, all of which were negative. These results were confirmed by our examination. The

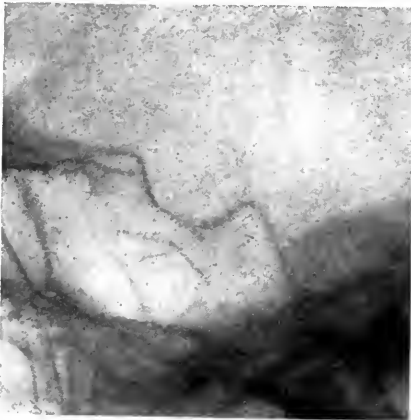
ILLUSTRATING DR. ZENTMAYER'S ARTICLE, "CONCERNING THE ETIOLOGY OF  
HEREDITARY OPTIC NERVE ATROPHY."



CASE 1.



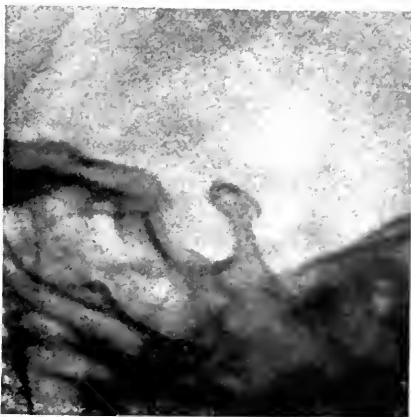
CASE 2.



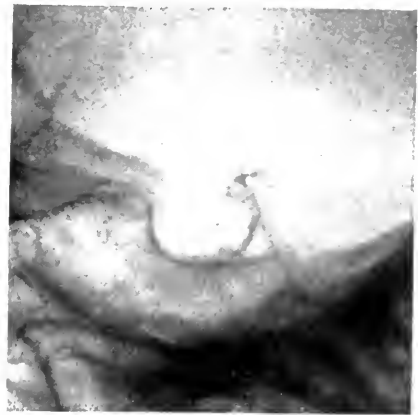
CASE 3.



CASE 4.



CASE 5.



CASE 6.

FIG. 3.—Cases 1, 2, 3, and 4 affected. Case 5, unaffected. Case 6, unaffected, but presenting indefinite pituitary symptoms.



nose, throat, and ears are normal. The Bárány test is normal. V. R. E. =  $\frac{1}{60}$ ; L. E. =  $\frac{2.5}{8}$ .

R. E. The optic papilla, which is atrophic and is of a greenish white hue, has a deep pit-like excavation. The perivascular lymph sheaths are full. The vessels are contracted and the arteries too bright.

L. E. The papilla is gray and the vessels of a better size than in the R. E.

The X-ray measurements of the pituitary fossa are anteroposterior diameter 13mm; depth, 12mm.

The opinion of Dr. Pancoast that in cases 1, 2, and 4 the pituitary fossa was either above the average normal or at the maximum normal was confirmed when the cases were shown before the Philadelphia Roentgen Ray Society, and it was suggested that other members of the family who were apparently unaffected be examined as controls. Accordingly one male and one female of the second family were X-rayed. The male (C. S.) was 20 years of age and showed no ocular disturbance other than a high hyperopia. The pituitary fossa measured 9mm anteroposterior diameter and 9mm deep. The fossa was therefore much smaller than in the two affected members.

The apparently unaffected female (M. S. E.) is 30 years of age. The fossa measured 12mm anteroposterior and 10mm deep and is therefore on the borderline of being abnormally large. She subsequently came to my office for an ocular examination being fearful of a fate like that of her brothers. She is married and has two living girls aged 8 and 9 years. The last two children were boys, the first dead born, the second living but eight hours. She wore glasses at 14 years of age for a period of one year. She complains of momentary obscuration of vision, a great deal of headache, and annoying sweating of the left side of the head. Corrected V. =  $\frac{5}{4}$  (normal).

The right papilla is normal, the left shows all but outer border obscured and slightly prominent. Central lymph sheath full. The visual fields showed decided concentric contraction for form and color with a minute central relative scotoma in the right field and an equally small paracentral relative scotoma in the left. Upon a second testing, five days later, on a Peter campimeter, no scotoma could be demon-

strated in the left field. She was examined neurologically by T. H. Weisenburg who found nothing abnormal aside from the left sided head sweating.

REPORT OF THE X-RAY EXAMINATION MADE BY DR. HENRY K. PANCOAST.

FAMILY C.: C. C.—At the first examination of this patient it was thought that the pituitary fossa was negative but after an examination of his brother, a re-examination convinced us that there was possibly a slight deepening of the pituitary fossa. This is about on the borderline of normal size.

J. C.—The pituitary fossa of this patient is slightly past the borderline of normal size and it is undoubtedly somewhat enlarged, both by deepening and in the anteroposterior direction.

REPORT OF THE X-RAY EXAMINATION OF FAMILY "S."

FAMILY S.: E. S.—The measurements of the pituitary fossa showed the fossa to be 13mm in the greatest anteroposterior diameter and 12mm deep.

F. S.—The pituitary fossa appeared to be on the borderline of normal. There is no evidence of any unusual pressure. The floor and posterior processes are thin but not out of proportion to the rest of the skull although thinner than in the case of the brother, C. S. The skulls of both C. S. and E. S. are of about the same thickness. The left anterior clinoid process is hollowed out beneath while the right is not. Both are normal but we usually do not find this combination. Measurements of the pituitary fossa—12mm anteroposterior diameter and 10mm deep.

C. S.—(One of the normal members of the family). The skull is thin. The pituitary fossa of normal size, and the processes about of normal thickness and apparently thicker than the two affected brothers. Measurements of the fossa, 9mm anteroposterior diameter and 9mm deep.

Mrs. M. S. E.—(Indefinite pituitary symptoms). The fossa in this case is large and on the borderline of being normal as in the case of F. S. The posterior clinoid processes and the floor are thinner than in the normal



brother. The skull is thicker than in any of the three brothers. Measurements of the fossa—*12mm* antero-posterior diameter and *10mm* deep.

Dr. Pancoast concludes that as each of the four cases showed a pituitary fossa which was either at the borderline of being abnormal in size or was slightly past this point, it would seem as though a pituitary enlargement might be regarded as a possible cause of the condition.

Concerning the X-ray findings in the female who certainly has not, at present, an optic atrophy, but who has subjective symptoms, ocular fundus and field changes suggestive of chiasmal disturbance, it would seem not unfair to hold that at the present time it cannot be said to refute Dr. Pancoast's views.

It may be recalled that recently Fisher brought forward the following facts from which he argues an inherited temporary disorder of the pituitary body as the primary cause of Leber's disease.

Leber noticed a great tendency for the visual defects to appear at or about the age of puberty, and that evidence of a neuropathic type was afforded by such symptoms as headache, vertigo, tremors, numbness of all the limbs, or even epileptic fits; that in several reported cases there were similar symmetrical field defects; that patients with Leber's disease, as also those with rapidly developing pituitary body growths, often complain of subjective phenomena of light and colors often as "seen through a blue mist"; that variation in the degree of central amblyopia which occurred in Leber's disease is more consistent with an outside influence on the visual pathways than with primary changes in the nerve fibers; that in both conditions there is an epochal relation between the onset and the period of puberty and the climacteric and that in both, frequently in early stages of the disease, a very mild papillitis can be detected. In one of two affected children of a family, roentgenoscopy of the skull was negative, while the other showed a cellular or honeycombed shadow in the depression of the sella turcica. That similar changes were not found in the two cases he attributes to the fact that the negative finding was in a case of two years' standing. As the visual

symptoms in Leber's disease are progressive up to a certain point and then come to rest, he argues that if the lesion which gives rise to these symptoms is due to some disorder of the pituitary body it also must needs be temporary and transient.

Recently Pallock reports 2 cases of hereditary optic nerve atrophy each showing a small bean-shaped shadow within but below the center of the sella.

In regards to heredity, Harvey Cushing remarks that there may be certain inherited deviations which may in all likelihood be attributable to transmissible ductless gland properties; and a functional glandular instability may exist in these individuals which makes them more susceptible, under stress, to alterations which border on the pathological.

It would seem to me, that our data are insufficient upon which to draw conclusions. Aside from the fact that the normal fossa must vary greatly in size and often overstep the limits set as maximum normal, our present knowledge of heredity in connection with disorders of the pituitary body is too vague to permit of assertive statements.

I wish to express my thanks to Dr. Barry for his kindness in permitting me to publish the notes of the second family.

REPORT OF THE TRANSACTIONS OF THE SECTION  
ON OPHTHALMOLOGY OF THE ROYAL  
SOCIETY OF MEDICINE.

BY MR. HENRY DICKINSON, LONDON.

An ordinary meeting of the Section took place on June 12th. Mr. WILLIAM LANG, F.R.C.S., President, occupying the chair.

The President first referred to the decease of Mr. John Couper, a colleague with whom he had long been associated at Moorfields Hospital. He considered that Mr. Couper's chief claim to distinction was his special ophthalmoscope for estimating refraction by the direct method. It was this which made possible the late Márcus Gunn's minute studies, a work in which Mr. Gunn was worthily followed by Mr. Bardsley.

Dr. G. H. GOLDSMITH exhibited a case of **cystic swelling of the disk**. On the left side there was a large coloboma of iris and choroid. The disk was almost completely eclipsed by a swelling which he adjudged to be cystic. It was semi-transparent, and over the swelling could be seen two vessels.

Mr. F. A. C. TYRRELL showed a case of **Mooren's ulcer**, in which the corneal denudation was extensive. About a year ago, the patient attended St. Mary's Hospital with a large denuded area, the only epithelium being 4mm in the center of the cornea. As two attempts at cauterizing did not result in healing, he employed the conjunctival flap method, using purse-string sutures. The conjunctiva adhered to the denuded area in the outer part, and the cornea vascularized, but the case did not progress very well. At the end of a month, he determined to unite the lids in the center, in order to be able to flush the conjunctival cavity through from side to side. The cornea was now clearing up, and the patient could now

see fingers at the distance of a meter, and the pupil and iris could be seen.

The President said that in one case he touched the cornea with solid flavine, and though the patient complained of pain, it was very successful. Weaker applications were generally useful, but this was a more stubborn case. |

Mr. LESLIE PATON, who had watched Mr. Tyrrell's case, confirmed its progressive character before treatment. It was the experience which had been gained in connection with sewing the lids in neuropathic keratitis which led to that method being adopted here. Clearing of the cornea followed equally well in cases of herpes in which the ulceration was deep. In the keratitis following operations on the Gasserian ganglion, however, the subsequent separation of the lids led, in his experience, to a relapse, unless at least a bridge of tissue of at least 1 mm were left. |

Mr. J. B. LAWFORD said his experience differed from Mr. Paton's in this respect, for a colleague of his at the hospital did a number of Gasserian ganglion operations, and Mr. Lawford sewed the lids together, and in not one of them was there a relapse when the lids were separated at a subsequent date.

Mr. A. ROXBURGH showed an **implantation cyst**. The patient, a girl æt. 14 years, had her eye struck by a tin toy aeroplane. The mother stated that the eye was operated upon immediately afterwards—probably at that procedure the prolapsed iris was removed. A fortnight ago the girl complained of pain, pricking, and headache. The mother then noticed a dark spot in front of the eye. Vision had fallen from  $\frac{3}{4}$  to  $\frac{1}{4}$ , tension was slightly raised, but in the left cornea some iris was adherent to the back of the cornea. Beneath the cornea was a translucent cyst, about  $4\frac{1}{2}$  mm wide, which appeared to be embedded in the iris, looking in every respect like an implantation cyst, and it was increasing in size. He felt it to be his duty to tackle it, but as he had not had such a case before, he asked for advice.

Mr. TREACHER COLLINS agreed with the diagnosis, and called attention to the importance of the cyst being removed, otherwise it would certainly fill the anterior chamber, and produce an increase of tension. The tissue removed at the

operation should include the portion of iris forming the back of the growth; if that were left, it would make practically certain subsequent extension.

The President agreed, remarking that these cysts often proved to be more extensive than was at first supposed.

Mr. M. S. MAYOU exhibited, on behalf of Captain Moxon, luminous test-types, arranged on the principle of the luminous wrist-watch, but of course with a special arrangement of lettering. An ordinary person in a completely darkened room could, after getting accustomed to the darkness, see these letters at a distance of four feet. Mr. Mayou had ascertained that the paint used for the purpose consisted of radium bromide and zinc sulphide, the light being produced by the bombardment of the zinc sulphide by the radium. It could not, however, very well be regarded as a standard, seeing that the degree of luminosity varied with the age of the paint, and with the thickness of the lettering employed. The scientific standard for estimating the light sense and the degree of night-blindness—which was the object of the device—would be a screen illuminated from behind, the observer wearing green glasses.

Mr. LESLIE PATON exhibited a patient in whom **chicken-pox was associated with neuroretinitis**. He had been able to find a record of only one other case of the kind, though neuroretinitis had occurred in association with poli-encephalitis. There had been nothing else amiss with the present patient, and his blindness came on suddenly on the third day of his other disease. The large central scotoma left was now gradually clearing up.

Mr. C. P. BARDSLEY read a paper on a **new form of bi-focal for myopes**. He said there existed at the present day a school of ophthalmologists who taught that in early life myopia should be fully corrected, the subject wearing glasses to keep this up, however high the degree might be. This school argued that high correction only turned the child or young adult having myopia into a person with normal vision, and that thereby the accommodation was called into play, as was the accommodation of the emmetrope or the hypermetrope. The same people stated that if this full correction did not take place, certain dire results would ensue; accommodative power

would be lost, and there would be rapidly progressive myopia. But there had been no evidence adduced in favor of the idea that lack of full correction of myopia caused an increase of myopia. The author had himself been myopic since early childhood, and there had been only a slight degree of increase in it since that date, and when he was well over 40 years of age his accommodation was found, at the Ophthalmological Congress, to be more powerful than any so far recorded by that body. He asked why myopes should be robbed of the privilege of reading without using their ciliary muscles. On looking down through the lower glass in the ordinary bi-focals, there was considerable distortion of the image. To overcome this, the author angled his bi-focals; they were made of separate pieces of glass, slightly beveled against one another where the pieces met. Next, he angled the circular bi-focals, and to overcome the defect in bringing the edges of the upper and lower glass into continuous contact, he ground a toric curve on the upper lens, and a double concave or convex on the lower lens. He detailed the advantages resulting from this improved kind of spectacles.

Mr. L. R. YEALLAND read a paper on **hysterical disorders of vision**, based upon his experiences as resident medical officer at the National Hospital for the Paralyzed, Queen Square. He said hysterical disorders of vision could be grouped, clinically, into two classes. The first of these were conditions in which the contraction of antagonists could be demonstrated, and the second, those in which such was not susceptible of proof or demonstration. By contraction of an antagonistic group of muscles he meant the contraction of a group which should be relaxed during the performance of a given movement. This contraction of antagonists could be demonstrated in blepharospasm, ptosis, and spasm of accommodation. In blepharospasm there was simultaneous contraction of the levator palpebræ superioris and of the orbicularis oculi; failure of relaxation of the last-named muscle prevented the opening of the eye, hence there was apparent loss of vision. The action of the antagonists in spasm of accommodation could be explained on the assumption that the suspensory ligament of the lens performed an opposite action to that performed by the ciliary muscle. The suspensory ligament, held normally in a

state of tension, was only altered by the contraction of the ciliary muscle. When the latter contracted, the ligament relaxed; when the ligament was in a state of tension the ciliary muscle was relaxed. The phenomenon of contraction of antagonist could not be demonstrated in such conditions as limitation of the visual fields and amblyopia, but in these conditions contraction of antagonists in some other part of the body could be observed. The treatment was one of suggestion, and must be got through at one sitting. It produced complete relief of the physical disability, though much could not be hoped for in the way of permanent improvement in the mental state.





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Statement of the ownership, management, circulation, etc., required by the act of August 24, 1912, of *The Archives of Ophthalmology*, published at New Rochelle, N. Y., for October 1, 1918.

*Editor*, ARNOLD KNAPP, 10 East 54th Street, New York City.

*Publishers*, G. P. PUTNAM'S SONS, New York City.

*Owner*, ARNOLD KNAPP, 10 East 54th Street, New York City.

Known bondholders, mortgagees, and other security holders, holding 1 per cent. or more of total amount of bonds, mortgages, or other securities, None.

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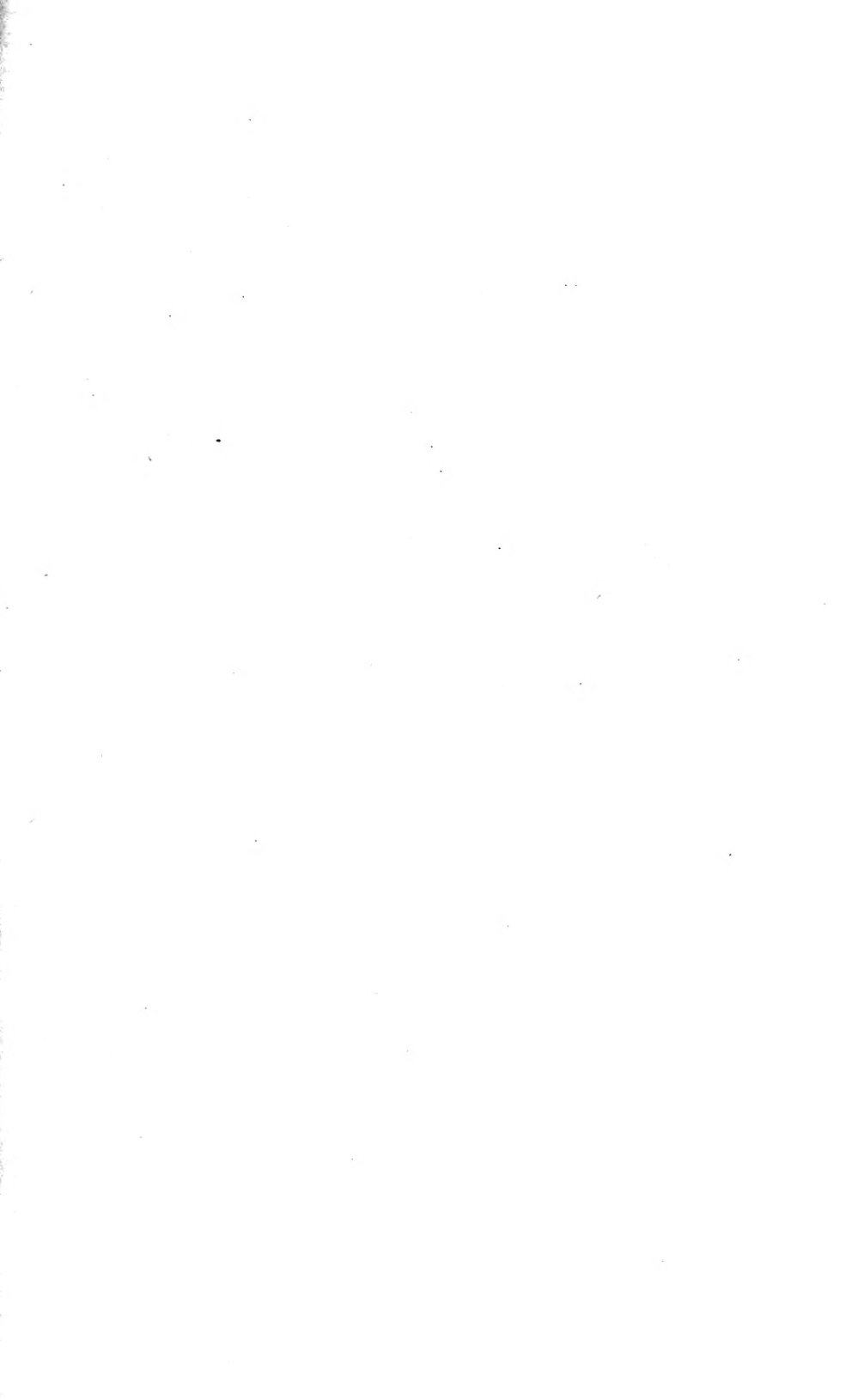
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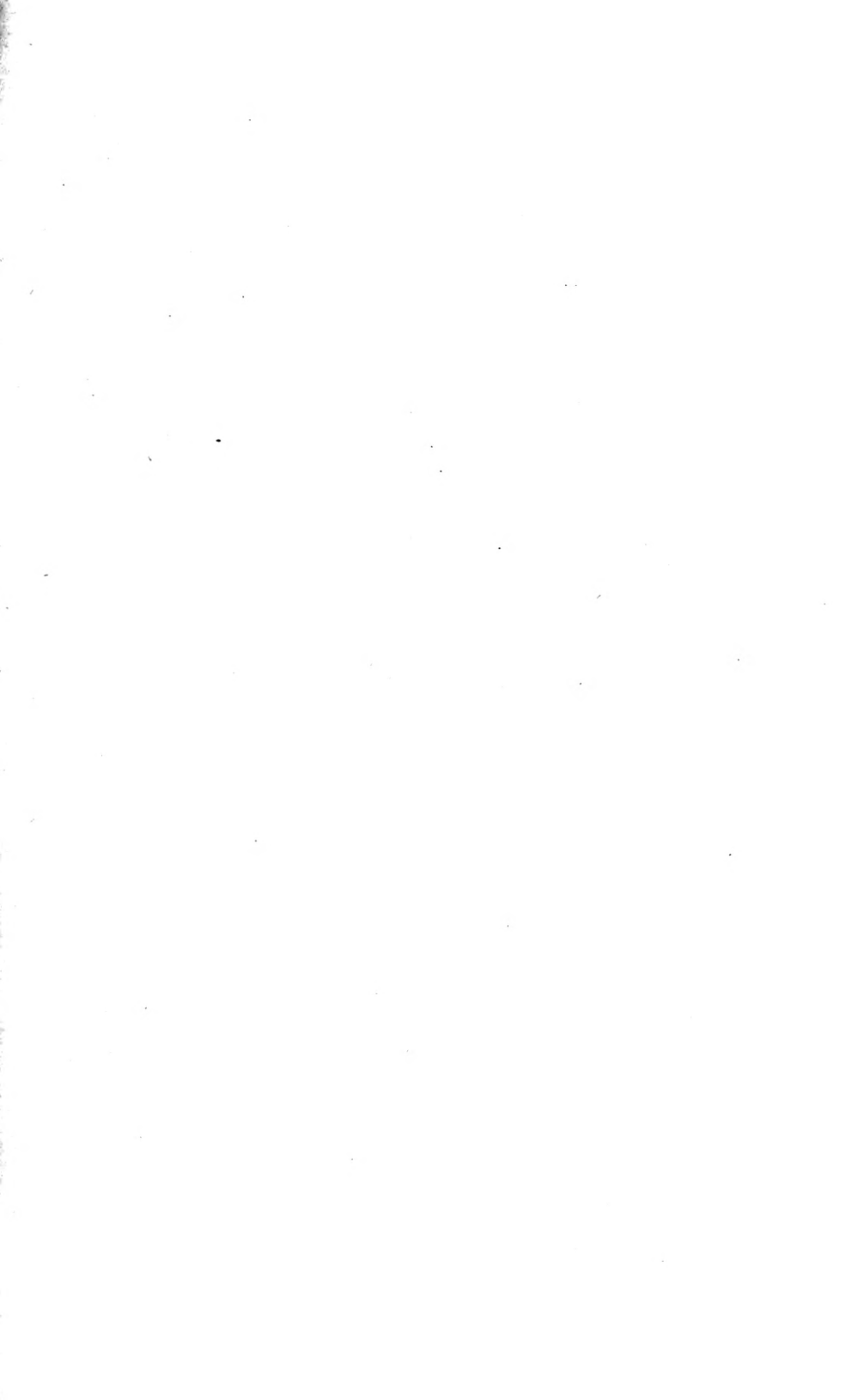


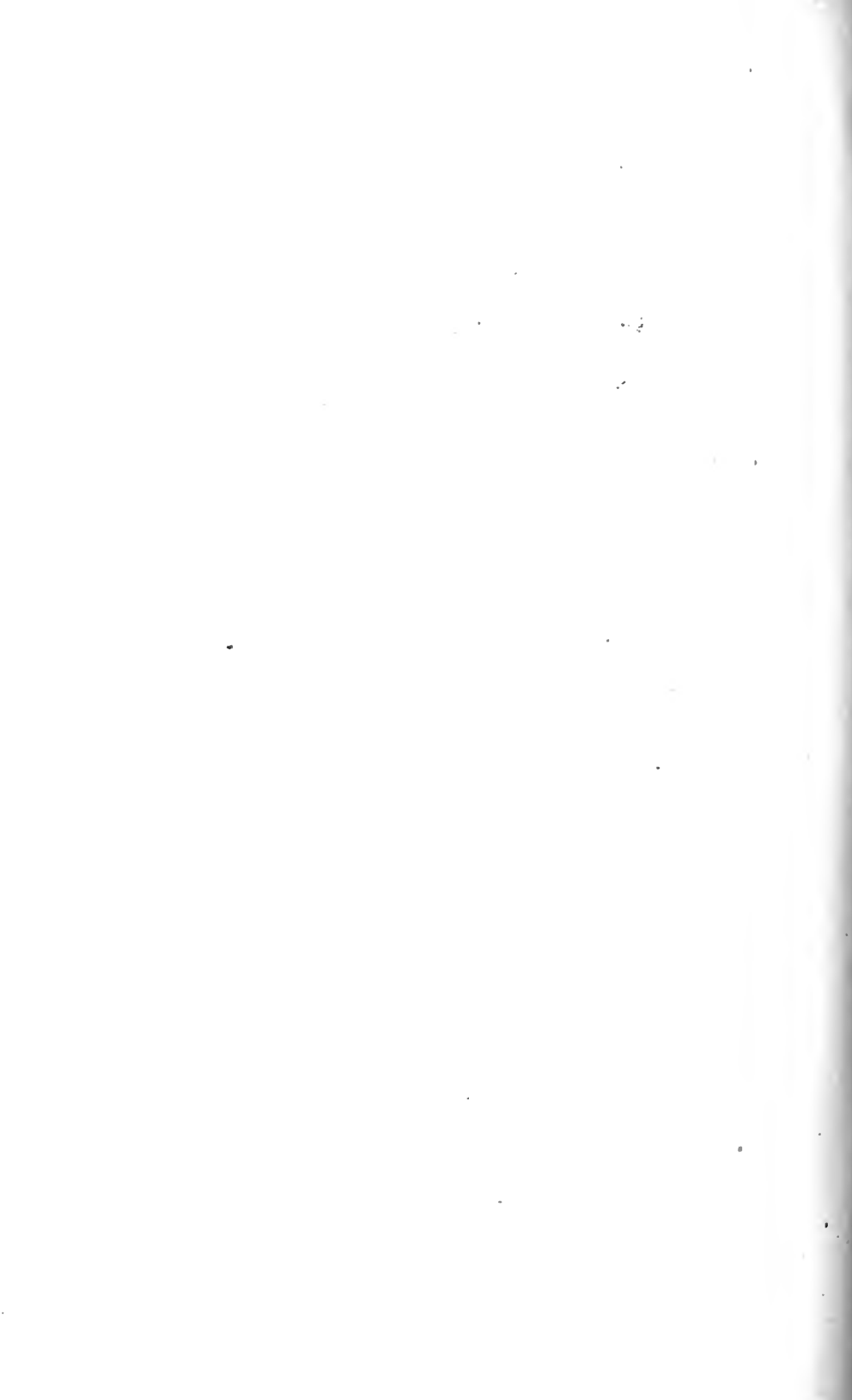












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